THE Journal Kansas Medical Society

OCTOBER
1955

VOL LVI
NO X

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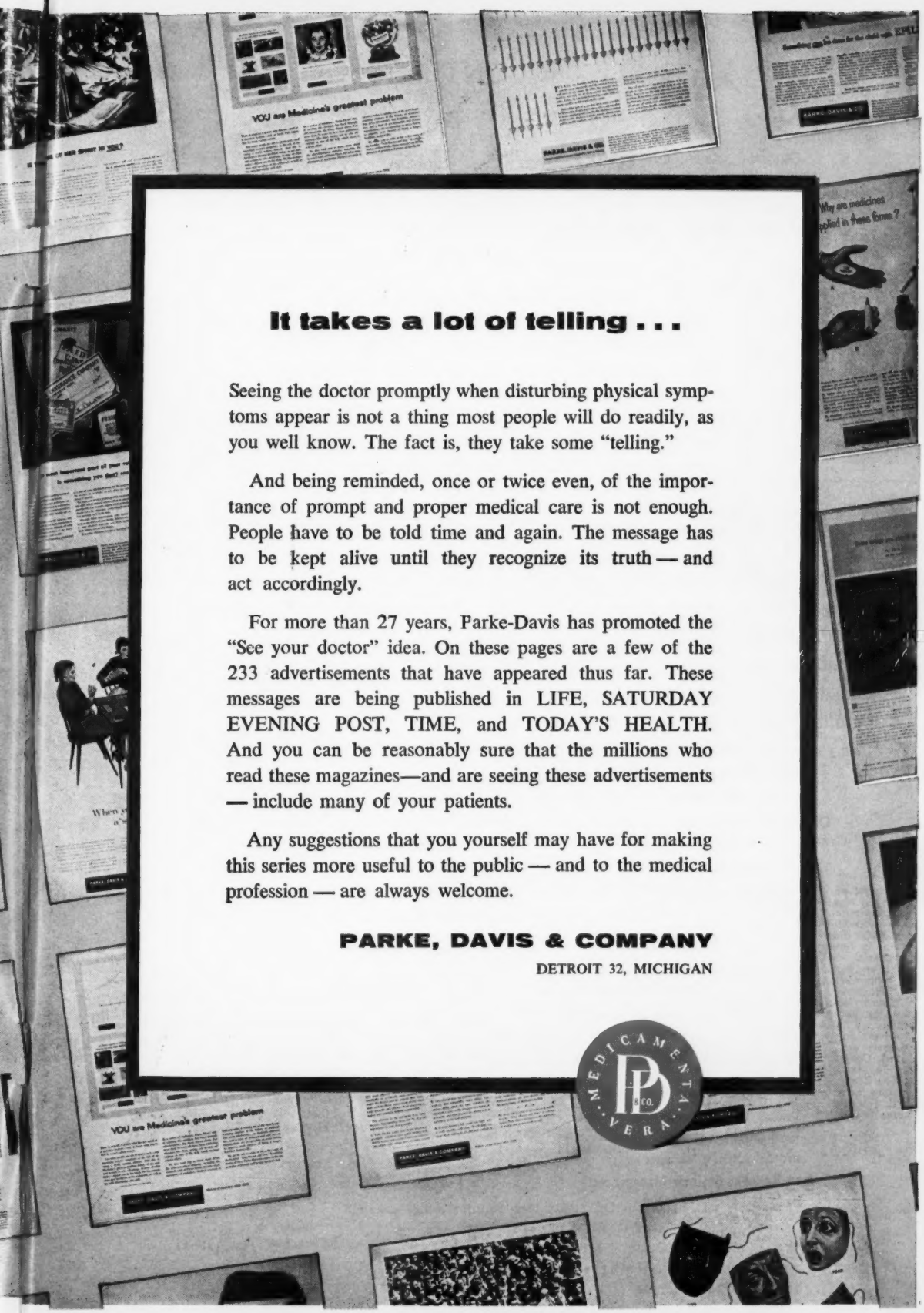

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TABLE OF CONTENTS

OCTOBER, 1955

ORIGINAL ARTICLES

Primary Uterine Suspension: A Surgeon's Views—Donald R. Davis, M.D., Mission, Kansas 547

Primary Uterine Suspension: A Gynecologist's Views—Dan L. Berger, M.D., Mission, Kansas 549

Present Status of Treatment of Advanced Laryngeal Cancer: Report of a Case—Paul Guggenheim, M.D., Topeka, Kansas . . 550

A Medical Student Looks at Blue Shield—Carl B. Younger, Los Angeles, California 554

EDITORIALS

Time May Have Been Cheated in an Untimely End 559

Cults 560

Porter Memorial 561

Our New President 564

MISCELLANEOUS

President's Page 558

Clinicopathological Conference 565

The Etiology of Diabetic Neuropathy—Senior Thesis 582

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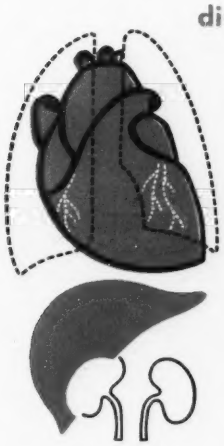

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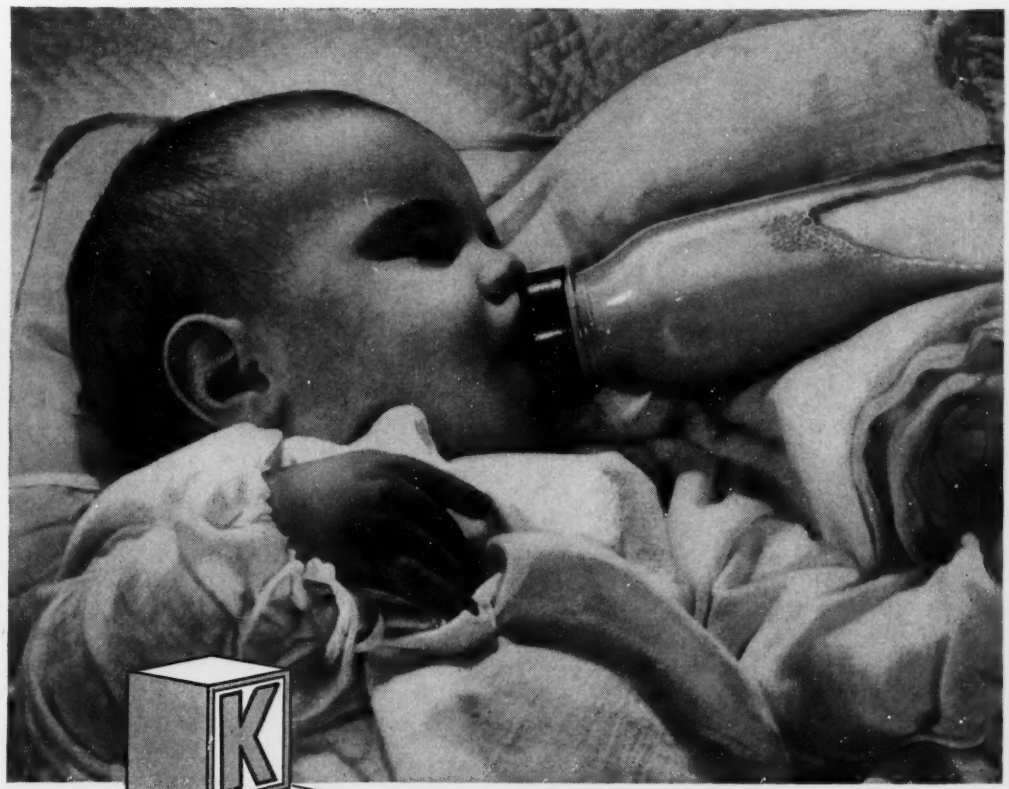

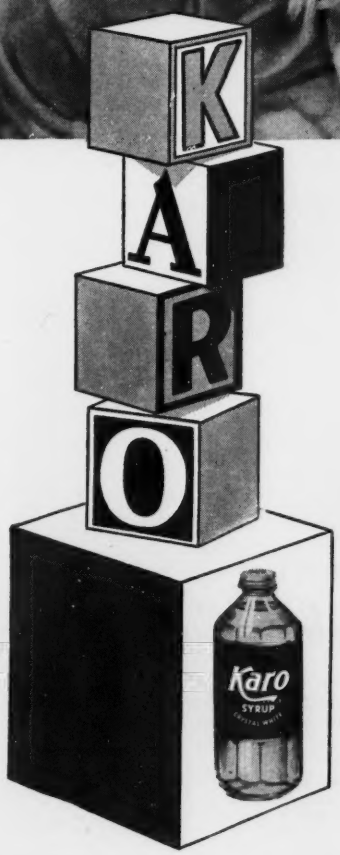

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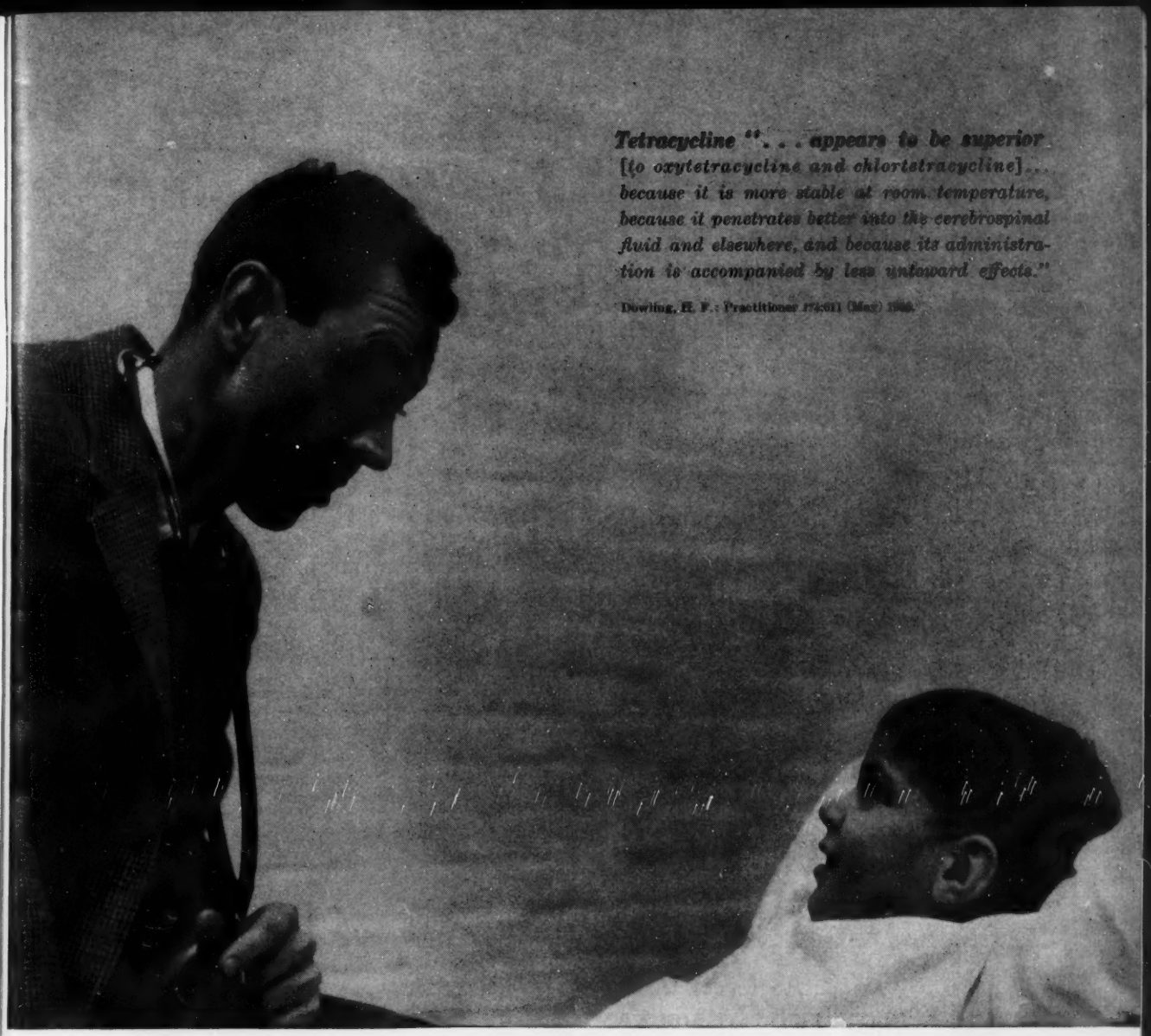
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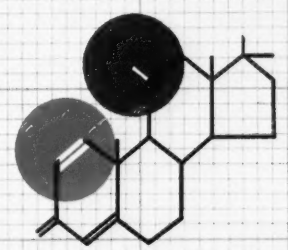

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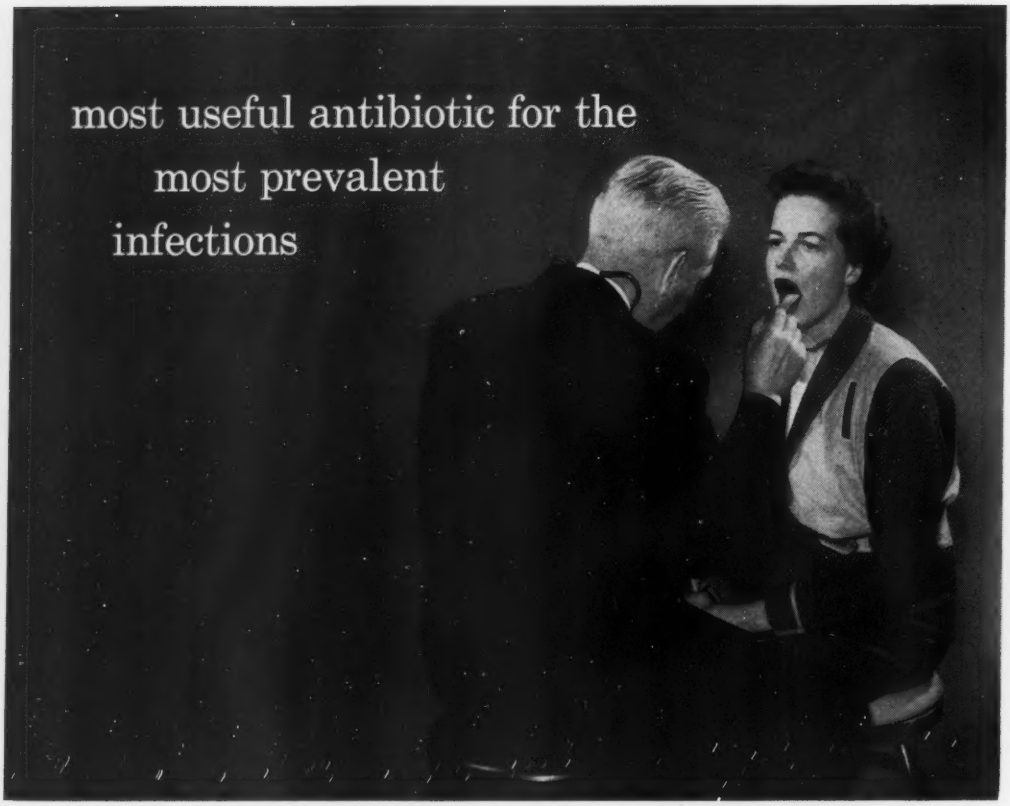

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Volume LVI OCTOBER, 1955 No. 10

Primary Uterine Suspension: A Surgeon's Views

Donald R. Davis, M.D.
Mission, Kansas

Transabdominal suspension of the uterus as a primary procedure is of questionable merit. Certain conditions attributable to laxity of uterine support, retrodisplacement of the uterus, prolapse uteri, and even proccidentia, have been treated frequently with operative procedures designed to return the uterus to normal intra-abdominal relationships. During recent years, however, the value of such an operative attack has been questioned seriously.

Although hysteropexy, or "uterine suspension," has been described in most leading textbooks of surgery, in comparison to other surgical diseases of the uterus it is interesting to note the relative sparsity of information available in surgical literature regarding the operation and its results. Also, it is of interest to note the incidence of primary hysteropexy appears to decrease inversely in proportion to the training of the operating surgeon. With regard to the high frequency with which this operation is encountered on operating schedules of our hospitals, it is noteworthy that hospitals possessing rigid educational requirements for membership to their surgical staffs have the lowest incidence of primary uterine suspension operations.

Opinions regarding primary hysteropexy are divided into two camps. There are those who are favorably impressed with results of uterine suspension and believe it to be a sound procedure based on definite indications. On the other hand, there are surgeons and gynecologists who believe the operation is worthless, unsound in principle, based on a battery of indefinite indications, and exposes the patient to needless surgery and anesthetic risk. Shamefully to say, there remains a small intermediate group devoid of definite individual opinion but who recommend and perform the operation, possibly in pursuit of a surgical fee!

To state that an operation designed to relieve the patient of symptoms is unsound and valueless is insufficient, in my opinion, unless such a statement can be based on a large series of case histories with corroborating follow-up data and controls. Conversely, to perform an operation which is of questionable value is equally ridiculous unless an objective effort is made to determine, from the results obtained, the true value of the procedure.

The nature and multiplicity of symptoms commonly attributed to retrodisplacement of the uterus, backache, heaviness in the pelvis, a sensation of bearing down, dysmenorrhea, menstrual irregularities, dyspareunia, recurrent endometritis, and infertility, make it extremely difficult to select a common denominator useful as criteria in such a study. Obviously, however, there is need for compilation and documentation of statistical data which will outline clearly the indications for primary hysteropexy or discredit the operation completely, whichever the case may be.

Retrodisplacement of the uterus, congenital or acquired, is not a disease and is seldom symptomatic per se, nor is it life-threatening! The normal uterus occupies a position of moderate anteflexion, lying in a midline supravaginal position with its long axis approximately 60° from the horizontal plane. Under normal conditions, the uterus may deviate considerably from its theoretically "normal position"; it has been said that approximately 20 per cent of normal uteri lie in some degree of posterior displacement.[1]

To define normal uterine position is necessary at an educational level, but it appears the academic definition has been too precise. Physicians, taught to explain symptoms on the basis of pathologic changes, too often find it convenient to associate certain symptoms to the displaced uterus. More often than not, the same symptoms are encountered in persons with normally positioned uteri who show no other identifying pathology. To assure a patient that hysteropexy will alleviate her symptoms under such circumstances is sheer guesswork and based on something other than scientific knowledge!

Prolapse of the uterus occurs as a result of relaxation of fibrous sheaths surrounding blood vessels and accompanying structures which supply the pelvic viscera, not as a result of inadequacy of the ligaments of the uterus and muscular action of the pelvic diaphragm. The round ligaments are vestigial structures, analagous to the gubernaculum testes, having functioned in dragging the ovaries from their embryonic positions on the genital ridge to their final resting place. The broad ligament, a true meso-ovarium and mesosalpinx, plays no role in mechanical support of the uterus. The uterosacral ligaments are simply folds of peritoneum, void of any major supportive function. The sole supporting element of the uterus is a perivascular fibrous investment, derived from the endopelvic fascia, accompanying blood vessels in the parametria, and firmly attached to the cervix.

Obviously, with due consideration to anatomical and physiological changes incidental to gestation, ligamentous suspension and ventral fixation of the uterus is a poor approach to the problem of prolapse uteri. Symptomatic prolapse requiring treatment during the child-bearing age may be adequately managed with an operation of the Fothergill type; after the child-bearing age, vaginal hysterectomy has much to recommend it.

I am of the opinion that indications for primary uterine suspension are exceedingly few but admit there are rare instances in which the operation may be of value. Perhaps the most feasible indication is the problem of long standing sterility where, due to retrodisplacement, the cervix lies in apposition with the anterior vaginal wall. Replacement of the uterus to normal relationships, temporary though it may be, will direct the cervix into the seminal pool in the posterior fornix. Candidates must be selected carefully, and the operation should not be done unless both husband and wife have been studied adequately. Furthermore, malposition must be persistent despite conservative measures, including attempts to replace the uterus by manual manipulation, trial by pessary, and exercises.

Those in disagreement with this indication maintain the same can be accomplished simply by having the patient sleep on her stomach after sexual intercourse. Such a simple solution may not be entirely adequate in that it permits postural drainage of the vagina by reversing the vaginal axis.

A second conceivable indication for primary hysteropexy is severe dyspareunia associated with retrodisplacement of the uterus. Although the normal uterus is not a tender organ, even when it is malpositioned, persons with retrodisplacement occasionally complain of discomfort during sexual intercourse. The uterine corpus, lying posterior to the vaginal canal, may be impaled by the intruding penis with each masculine oscillation yielding a sensation of discomfort. Such appreciation of coital displeasure is undesirable and may contribute to frigidity problems endangering family integrity.

Secondary uterine suspension, an item not pertinent to this paper, is indicated where excessive operative denudation occurs with conservative operations for endometriosis associated with concomitant retrodisplacement of the uterus. The operation prevents posterior fixation of the organ and provides a method of peritonization. Also, whenever the adnexa has been sacrificed, it is a wise procedure to suspend the uterus.

I can conceive of no indication at all for ventral suspension. Should hysteropexy be indicated, primary or secondary, I believe a ligamentous suspension of the Gilliam or Baldy-Webster type to be the operation of choice.

SUMMARY AND CONCLUSIONS

1. Primary hysteropexy is of questionable value as a method of treating retrodisplacement of the uterus. The operation has no place whatever in the management of prolapse uteri or proccidentia.

2. There is need for documentation of statistical data which will delineate or deny the indications for primary hysteropexy.

3. Primary hysteropexy may be of value in sterility problems of long standing where the cervix lies in apposition with the anterior vaginal wall. Also, severe dyspareunia associated with retrodisplacement may be an indication for primary hysteropexy in rare instances.

4. It is the author's opinion that primary hysteropexy is performed more frequently than adequate indications would justify, and a certain portion serve only to facilitate amortization of the mortgage on the operating surgeon's Cadillac!

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Primary Uterine Suspension: A Gynecologist's Views

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Thirty years ago Arthur Dean Bevan[1] editorialized ". . . We must conclude without any reserve that the time has arrived when operations done on women for retroposition of the uterus, for this condition alone, are unwarranted, unnecessary, and indefensible because of two facts: That these operations are of no benefit to the patient and they carry with them the serious risks such as I found in the three cases which I have enumerated." That this and a hoard of similar utterings have been disregarded in the ensuing years is evident to anyone casually scanning the operative schedules in a majority of our hospitals today.

The uterus is normally suspended by a sling of fascial tissues (the cardinal ligaments or Mackenrodt's ligaments) which are condensations of fibrous tissue lying in the base of the broad ligament and attached to either side of the cervix about the level of the internal os. This has been adequately demonstrated by Mengert[2] and others.

The usual position of the uterus is in anteversion and slight anteflexion. The normal uterus is an organ of great mobility, and only about 80 per cent of uteri are so placed.[3] A movable uterus which is retroverted and retroflexed is no more abnormal than a hand that is temporarily held above the head, even though it may deviate from the classical anatomical position.[4] Operative procedures on the approximately 20 per cent of normally retrodisplaced uteri are entirely unjustified.

The problem of prolapse of the uterus is in fact a multifaceted one which is composed primarily of two factors: relaxation of the normal supporting structures (cardinal ligaments), and elongation of the cervix itself. Retrodisplacements have been implicated, but the cause and effect relationships are not entirely clear. A high percentage of nulliparous women with prolapse have an occult spina bifida which has led some observers to postulate a neurogenic influence.

The use of any of the suspension operations totally ignores the primary pathology of the condition, and ventrofixation is merely a stop-gap procedure. The pathology remains and continues to progress with the result that the cervix gets even longer and the uterus itself begins to elongate from its attachment to the abdominal wall. This is a gradual process taking up to several more years to reach its former extent of descensus.

Hysteropexy is thus seen to be an irrational approach to the problem of prolapse and one which will be ultimately unsuccessful in a high proportion of cases. Its use in elderly women who must undergo laparotomy for other reasons is justified occasionally, but as a primary procedure one of the several vaginal operations carries greater chance for success and, in general, a lesser mortality.

Retrodisplacement of the uterus has been blamed for a wide variety of pelvic symptomatology varying from pain to leukorrhea. In a *very small* group of each of these conditions it probably does play a part. It can be stated categorically, however, that the movable retrodisplaced uterus is not a pathological uterus and that other causes of pelvic pathology must be excluded.

The uterus fixed in retroversion is held there either by adhesions from a previous inflammatory disease or pelvic surgery, or by endometriosis, and the symptoms are caused by this pathology and not the retrodisplacement. Peritoneal adhesions from a suspension or other laparotomy may only increase the symptoms or introduce new ones. The amelioration of symptoms one often sees with suspension of the retrodisplaced uterus is temporary or incomplete in a high percentage of cases, and the fleeting good results are due in large part to the placebo effect attendant on any therapeutic regime, plus a period of bed rest with release from physical and emotional stress.

The *rare* case of retrodisplacement that will benefit from hysteropexy must be carefully selected if we will avoid unnecessary surgery. If a uterus can be replaced bimanually and held there with a pessary with complete relief of symptoms, if these symptoms recur with removal of the pessary and subsequent retroflexion of the uterus, and can again be banished by anteversion of the uterus and a pessary, then, and only then, may a patient be properly considered for hysteropexy. Again we must bear fully in mind the placebo effect of merely inserting a pessary when we come to the final preoperative evaluation of these patients.

There are many other methods of dealing with patients with prolapse and retrodisplacement which may yield permanent or long lasting cures and certainly are sufficient for a large number of patients. Such procedures include Kegel[5] exercises, positional exercises such as knee chest position and sleeping on the stomach, and even the permanent use of a pessary. This last may well be the best procedure in elderly, poor risk patients who are nearing the end of their life span or as a temporary measure in younger women who desire another child. These lack the ad-

vantage to the surgeon of showmanship and a surgical fee, but to the patient they may mean the difference between a reasonably comfortable life and a fatal operating room accident.

Hysteropexy as an operation should not be totally cast aside, but rather it should be viewed in its proper relationship to pelvic surgery as a whole. If a laparotomy is indicated purely for other reasons and a fixed retrodisplacement is encountered incidentally, then certainly the operator is justified in freeing the uterus and holding it in an anterior position.

If the patient has had an adequate trial with a pessary, as described, and fulfills *all* criteria, then a hysteropexy may be considered. The occasional elderly poor risk patient with prolapse whose abdomen must be entered for other reasons may well benefit from a ventrofixation, and it conceivably may hold for the rest of her short life. These indications are met only rarely and in few of them is hysteropexy the primary procedure.

SUMMARY AND CONCLUSIONS

The rationale of hysteropexy has been discussed. This operation as a primary procedure has been viewed with increasing disfavor by a vast majority of gynecologists and in some institutions has been entirely abandoned. The author feels that primary hysteropexy is rarely indicated and that its chief value is as an adjunctive procedure when laparotomy must be done purely for other indications.

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Present Status of Treatment of Advanced Laryngeal Cancer: Report of a Case

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There is little disagreement today about treatment of cancer limited to one vocal cord, uninvolved at either end and freely movable. Laryngofissure and x-ray therapy both give high cure rates (85 per cent or better). When a lesion exceeds this extent, the issue becomes considerably more complicated.

In moderately far-advanced lesions not amenable to simple laryngofissure (clipping operation), one must choose between radiation therapy, partial or total laryngectomy. In the past, statistical data of cures have not been especially helpful for two reasons: (1) the criteria for selection of cases for any given treatment modality varied widely from one clinic to another and (2) treatment methods, both radiologic and surgical, have improved so much in the past few years that presently available figures for five-year survival are for the most part based upon outmoded procedures.

Del Regato[1] has mentioned that few radiologists about the country were trained to treat laryngeal carcinoma. The radiologist either had to be well trained in the medical aspects of laryngology or else work in intimate association with a laryngologist. Most laryngologists, unfamiliar with radiologic methods and discouraged by earlier poor results with x-radiation, preferred surgical procedures, and only in a few centers did the necessary teamwork develop.

There is now general agreement[1] among laryngologists and conservative radiologists that x-radiation has little or nothing to offer in lesions which are either retrocricoid, subglottic, or sufficiently infiltrative to interfere with the motility of a vocal cord. Extensive lesions involving the aryepiglottic fold, pyriform sinus, or lateral hypopharyngeal wall, which used to be treated exclusively by x-ray with disappointing results, are now being salvaged by modern surgical methods. Radiologists,[2] here, are well content to be "second-wave assault troops," preferring the surgeon to remove as much involved tissue as possible to prepare the way for a maximally aggressive radiologic attack upon the disease without having to fear the miserable complication of radio-

chondronecrosis with its attending infection and sloughing.

Statistical results, fortunately, do not afford the sole avenue of investigation of the problems of laryngeal cancer. In fact, even good statistics of any large series of cases have, in general, "low resolving power" for analysis of many important therapeutic problems. No less scientific, and considerably higher in "resolving power," is the detailed clinical and pathological study of individual cases, sufficient in number to represent the characteristic clinical problems. For such study the clinician must devote considerable time to the detailed dissection of surgical specimens, a task which should not be entirely delegated to the pathologist.

Pathologic studies in various clinics[3, 4, 5, 6, 7] have shown that 25 to 30 per cent of laryngeal cancers have metastasized to the cervical lymph nodes when these nodes are not clinically palpable nor even grossly demonstrable at surgery. This gives an approximate figure for the failure rate to be expected from total laryngectomy in cases formerly thought suitable for this operation.

More than a decade ago, the old fashioned "narrow field" or "subperichondrial resection" of the larynx was replaced by a so-called "wide field" procedure in which at least the sternothyroid and thyrohyoid muscles were removed along with the larynx. The more recent pathologic studies have shown microscopic invasion of the *outer* strap muscles and other fairly remote structures in cases thought curable by the old wide-field method.

In the past few years the attitude of surgeons has changed considerably as a result of these observations, and it may be expressed thus: *Once the decision has been made to sacrifice the larynx, it is advisable to sacrifice with it as much, rather than as little, neighboring tissue as possible.* The tissues neighboring the larynx are, for the most part, not vital, and their removal entails nothing in the way of added disability while it does insure a considerable added margin of safety.

Within the past couple of years it has gradually become an accepted practice to perform simultaneous prophylactic radical neck dissection on the side of major involvement whenever the larynx is removed, a procedure amply justified by subsequent development of metastases in 30 per cent of cases when this is not done. The expectant policy of waiting until clinical metastases develop is going by the board. Bocca[7] said in 1953: "It is surprising that laryngologists should keep on arguing about a problem which has long been unanimously settled in other fields of cancer surgery." Very few figures are presently available for results of simultaneous laryngectomy-neck dissection. Justification must rest on sound pathological principles until fortified with definitive statistics which should be available within the next few years.

Modern laryngectomy requires meticulous anatomic workmanship. As a result of improvements in the techniques of replacement of fluid, electrolyte, and blood losses, and the employment of pentothal sodium supplemented by nitrous oxide, laryngectomy-neck dissection and other radical head and neck procedures can be carried out without haste and with a remarkable absence of shock and other complications.

The usual T-shaped incision has given way to the original U-shaped Gluck-Sorensen incision which parallels the sternocleidomastoid muscles, crossing the neck just below the first tracheal ring. This incision, recently revived by Ogura,[4] permits ideal access to both sides of the neck for purposes of exploration and radical dissection.

The modern operation encompasses removal of the larynx with all strap muscles, the hyoid bone, epiglottis, pre-epiglottic space, and one or more tracheal rings, plus en bloc neck dissection on the side of major involvement. This basic procedure is capable of extension to include resection of the base of the tongue, pharyngeal wall, cervical esophagus, or bilateral neck dissection. When reconstruction of the cervical esophagus is necessary, a flap hinged on one side is employed instead of the usual U-shaped flap hinged superiorly at the hyoid level. This lateral flap is used for purposes of esophageal reconstruction (Wookie operation).

In the last few years, the operation of partial laryngectomy (sometimes miscalled hemilaryngectomy) has been pushed to "extreme limits of usefulness." This category of procedures entails a laryngo-fissure-type approach in which, however, the larynx is not split in the midline as in the clipping operation, but is entered through the thyroid ala on the side of lesser involvement, well beyond this involvement (at least 1 cm.). Parts of one or both thyroid alae are resected. The patient is left with some kind of functioning larynx and does not face life with an anatomic divorce of his breathing and nutritive functions in the form of a permanent tracheostomy.

Some years ago, Hayes Martin[8] warned that attempts to extend the upper limits of usefulness of this procedure would result in a deterioration of statistical results, since cases would be so treated which might better have been subjected to total laryngectomy. It is interesting to observe that partial laryngectomy for extensive lesions is practiced mostly by surgeons who devote much of their effort to plastic work. For example, Pressman[9] speaks of disassembling and reassembling the larynx as one does a nose in performing rhinoplasty. Such a concept, if taken quite literally, would seem at variance with the basic oncologic principle of wide en bloc resection. (On

the other hand Ogura,[10] also an expert in rhinoplasty, states that his experience of rhinoplasty has contributed greatly to his technical facility of performing *radical* laryngeal surgery entirely by scalpel dissection.)

Conservative laryngologists interpret the indications for partial laryngectomy quite rigorously. Clerf[5] has decried the tendency to extend the procedure to doubtful cases and has pointed out that cicatrization of the larynx all too frequently results—even in the best hands—which makes early detection of recurrences difficult.

One wonders whether employment of partial laryngectomy for large, advanced intrinsic lesions really constitutes an "advance in technique" as claimed by some. The situation in regard to partial laryngectomy presents some analogies to surgery for cancer of the rectum. Here, a few advocate a "pull through" type of operation with preservation of the anal sphincter. Despite the fact that patients are just as reluctant to part with the not-uniquely-human function of defecation per rectum as they are to part with laryngeal speech, the vast majority of general surgeons unhesitatingly close forever the southern portal of the anatomy and leave their patients to face life with permanent colostomies. They have, for years taught that "It's better to be resectable than respectable." The laryngologist has somewhat more leeway in electing a definitive procedure. Though he may be conservative by nature, spending most of his time with the "smaller things of otolaryngology"—adenoids and tonsils, sinusitis, and allergy—he does well to emulate the courage of his general surgical colleague when the occasion arises.

In deciding upon the relative merits of total versus partial laryngectomy in each case, the clinician must keep in mind that his first duty is to save life, his second to preserve function. The incompatibilities of these two aims are logically quite complex in varying situations. No patient, even when fully informed of the relevant facts, is capable of making a rational choice in these matters. The clinician is wise who thinks the issue through to a decision without inviting the patient to participate directly or covertly in his deliberations.

Once a definite recommendation has been made, the patient may elect not to follow it, and the clinician is still free to suggest with a clear conscience the second best treatment. If given the opportunity by participating in the decision before the clinician is himself clear as to the indications, patients will almost invariably elect to risk their best chance of cure in favor of temporizing measures which will permit salvage of the larynx. The threat of death from the disease itself is less real to them than the more immediate one of surgical mutilation.

The clinician owes any patient in whom he thinks laryngectomy indicated, a thorough briefing on the prospects of a laryngectomized life. Fortunately, there is a great deal to be said on the credit side. The patient may be assured that the loss of speech function is only a temporary one, since the vast majority can be taught buccoesophageal speech in six weeks to three months. The very few who will not or cannot learn it can still obtain satisfactory results with the artificial larynx. The buccoesophageal voice, it is true, is usually somewhat flat and monotonous in quality but serves admirably to communicate and is readily understood over the telephone.

McCall[11] actually advocates the institution of buccoesophageal speech training before laryngectomy. Some training in the technique of belching is generally advocated preoperatively. Beyond this, however, in addition to the technical problems involved, it seems psychologically necessary for the threatened loss of the larynx to become a reality to which the patient then has an opportunity to react with appropriate psychic shock and ensuing depression, just as Cholden[12] has shown inevitably to occur in the newly blinded. This is a reaction similar to mourning the loss of a loved one, a necessary prelude to rehabilitation. The physician assumes here a supportive role without attempting the impossible as well as undesirable task of preventing altogether these psychic reactions. After rehabilitation is complete, a kind of compensatory euphoria becomes a more or less permanent feature of the patient's personality. Clerf[5] states that "the rehabilitated laryngectomized patient usually becomes an extrovert and exhibitionist and not only does his job well but literally becomes his brother's keeper. I have never seen more enthusiastic people in my life."

It should be remembered that the larynx is not, par excellence, the organ of speech, but only the housing of the vibrating reeds. Exquisite as is the cerebral control of laryngeal function, far more innervation is required to control the *articulating* mechanism of tongue and pharynx. Laryngectomy does not, therefore, rob the patient of the essentials of the uniquely human function of communication by articulate speech. He must learn to use a different vibratory mechanism which requires a new set of neuromuscular patterns in breath control and swallowing. It is worthy of mention that removal of the epiglottis and hyoid bone results in a firmer and more satisfactorily vibrating hypopharyngeal scar than is obtained by preserving these structures.

CASE REPORT

The following case report illustrates many of the problems discussed above.

This 55-year-old man first noted a "drawing sensa-

tion in the muscles of the left side of the throat" and pain in his palate on swallowing in 1949. This became worse in 1951, the patient becoming hoarse around Christmas time. Hoarseness was at first intermittent and associated with colds, but it gradually became worse. Saliva which the patient could not expel collected in the hypopharynx. Drinking hot fluids afforded some relief. The patient saw a laryngologist who was unable to view the larynx adequately with a mirror. The patient declined direct laryngoscopy. During the ensuing six months he became gradually worse, suffering from frequent choking spells which eventually brought him to me.

Past history revealed that prior to the present illness the patient had "never been sick for a day" in his life. He had been a truck driver for 21 years. He drank heavily and had smoked two to three packs of cigarettes daily since the age of six.

He was a ruddy-faced, well preserved man about six feet three inches in height. Weight had fallen from an average 235 to 208¼ pounds. His neck was thick and short. His voice was weak and hoarse. The nose was obstructed by a severe right sided septal deviation. The mouth was edentulous.

The larynx presented a dirty, greyish, ragged lesion involving the full length of the left true vocal cord. Anteriorly, the tumor crossed the commissure slightly; posteriorly, it involved the arytenoid. Superiorly, all of the left false cord and part of the aryepiglottic fold were involved. Despite the great surface extent of the tumor, the left vocal cord was freely movable. This was the more surprising in view of the fact that laminography of the larynx showed a large tumor mass (the size of a prune) occupying the left hemilarynx and completely obliterating the left laryngeal ventricle. No lymph nodes were clinically palpable in the neck. The remainder of the examination was negative. Biopsy showed squamous carcinoma, grade II.

On July 12, 1952, the patient was given a bilateral deep cervical nerve block, and a U-shaped mastoid-to-mastoid flap which included the platysma was dissected up to the under edge of the mandible. An endotracheal cuff was introduced through an incision in the cricothyroid membrane, and the patient was put to sleep with intravenous pentothal sodium which was used exclusively for the remainder of the operation.

On exploration of the left neck, a group of enlarged, firm nodes was found along the mid-portion of the internal jugular vein. (It will be recalled that these nodes were not clinically palpable.) An extensive resection was carried out, which included the larynx with epiglottis and half the vallecula, all strap muscles, the entire hyoid bone, and the entire left pyriform sinus (including its lateral wall) en bloc with a left radical neck dissection. The internal jugular vein before ligation measured an inch and a quarter in diameter.

After inserting a Levine tube, every available scrap of tissue—inferior constrictor, fascia, etc., was utilized to reinforce the esophageal closure. The thyroid gland was divided in mid-isthmus and the halves everted and sutured to the carotid sheaths to make room for a large tracheostome. Around the stoma, skin and tracheal mucosa were united by some 40-odd fine silk sutures placed 1 mm. apart. An 18 French catheter was placed in each carotid gutter and a number 12 laryngectomy tube inserted in the stoma. The patient received five pints of blood and 2500 cc. of intravenous fluids.

On the first postoperative day the patient walked to the bathroom. In addition to daily intramuscular penicillin, the catheters in the neck were irrigated twice daily with 250 cc. normal saline containing 400,000 units of penicillin. This was followed by one half hour of hot compresses to the neck. Esophageal fistula occurred despite the care exercised in closing the wound.

On July 16, the patient developed a slight left-sided surgical mumps. Penicillin was replaced by aureomycin, and three x-ray treatments were given over the left parotid gland. The laryngectomy tube was removed on July 22. By August 18, Dakin's solution introduced through the catheters in the carotid gutters could no longer be tasted, and clear fluids by mouth were begun without leakage. The patient was discharged from the hospital on August 20, and an intensive course of deep x-ray therapy was undertaken by Dr. James Kelly, Sr., of Omaha.

In December 1952, the patient developed a small fistula on the left lateral aspect of the neck which, on biopsy, showed only necrosis but no carcinoma. Healing occurred rapidly beneath a simple dressing of bacitracin. The patient then developed a tracheitis sicca which necessitated a number of bronchoscopies for removal of crusts. It was found useful to dry the trachea with cotton and then directly apply lanolin ointment to it. In addition, the patient's quarters were humidified as much as possible.

Detailed description of the surgical specimen will be omitted for the sake of brevity. Suffice it to say that carcinoma had invaded the pre-epiglottic fat. It had also penetrated the left aryepiglottic fold and was on the point of rupturing into the left pyriform sinus. There was widespread cervical node involvement on the left. Histologically, the lesion was squamous carcinoma showing acanthosis and pearl formation, with more anaplasia in the deeper layers.

Some psychologic observations may not be amiss here. This patient understood nothing of the art of being sick, never having previously experienced sick-

ness. He was in serious marital difficulties and was depressed over them. After his recovery from the operation, he told the physician that he had neither expected nor hoped to survive it. He adamantly rejected all efforts to teach him to use the buccoesophageal voice. He would grimace and gesticulate wildly in an effort to make himself understood without speech, and end by angrily pencilling a scarcely legible note. After a year, he reluctantly accepted an artificial larynx which he used as little as possible. In the spring of 1953, the patient and his wife were divorced.

A year later, in June 1954, the patient was hospitalized at the Veterans Administration Hospital in Lincoln, Nebraska, remaining for three months for speech training at the University of Nebraska Speech and Hearing Clinic. He made fair progress. During this hospitalization, an indurated, slightly tender mass was noted in the left mastoid region. In July, this was surgically removed in Lincoln and found to be merely an old, organized hematoma. The patient was again hospitalized for further speech training from September, 1954 to March, 1955. Since his discharge he has remained entirely well and is working at a sedentary occupation. Doubtless his depressed state prior to surgery interfered with the "mourning process" which should have followed it, and this fact drastically retarded his rehabilitation.

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A Medical Student Looks at Blue Shield

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As the physician of the future, the medical student of today looks at the present as he reads of the past and dreams of tomorrow. His observation of the present is preparation for his participation in the future. He relates his personal struggle to the strivings of the profession. Professional problems become personal problems. And he knows that when he becomes a functioning part of medicine, personal problems will then be professional problems. Therefore, he is more honest with himself if he analyzes a problem that will soon be his with respect for his own feelings instead of attempting to achieve objectivity through superficial elimination of subjectivity. It is with this attitude that this medical student looks at Blue Shield.

Medicine is a science of service. It was created by man, for man, to protect man's most precious possession, life. How to serve his fellow man most effectively is the universal challenge of medicine. It is the personal goal of the physician. In a democracy dedicated to the proposition that all men have the right to equal opportunity, the proportions of the challenge are enormous.

Accepting the challenge as a personal problem, the medical student asks of himself, how should this be met?

In answer, he focuses first on the primary participants in the practice of medicine, the physician and his patient. Their relationship is the essence of medicine.

He narrows his scope of observation and considers this relationship in terms of the patient. First and foremost, the patient has the right to medical care when he needs it. Neither the quantity nor the quality of care should be solely dependent on the patient's economic status. For this care to be fully effective, anxiety stress which can complicate or create illness must be kept to a minimum. Doubt in the doctor and concern over cost are frequent causes of preventable

This essay won second prize in a national contest sponsored by Blue Shield and the Student American Medical Association. Mr. Younger is a student at the University of California at Los Angeles School of Medicine.

anxiety. If the patient finds that he lacks faith in his physician, he should have the right to choose another in whom he can have confidence. He should be able to direct all of his energy toward getting well and not have it consumed by worry over the bill.

The primary responsibility for the physician-patient relationship rests with the physician. It is essential that he be able to concentrate his skill and concern on each patient as an individual. He must have the freedom to decide, on the basis of his knowledge and experience, how best to treat every person who comes to him for care. He must have the opportunity to establish the closeness necessary for communication. He must be in a position where he is free to serve the needs of the patient and practice in a manner that is satisfying to himself.

The relationship of physician and patient that is based on mutual respect benefits both and forms a solid foundation for sound medical practice.

This, then, is the answer for the student.

But how have others answered the question in practice?

Since early in the history of medicine post-paid private practice has been the dominant form of medical service, and it continues to the present day. This is a system whereby a patient chooses a doctor when in need of medical care. The care is prescribed for the patient by the physician. The patient is billed by the physician according to the services rendered. With complete freedom of choice, the patient may select a doctor in whom he has confidence. He has the right to solicit different or additional medical advice. These factors, however, are limited by the condition of his pocketbook. The request for needed medical care is frequently postponed or never made because the patient may feel that it is easier to be sick than to worry about the doctor bill. His freedom of choice is limited by his income tax bracket. He may lack confidence in his doctor because he *can* afford him. If more help or treatment is indicated, he can get it *if* he can pay more for it.

The core of private practice is the patient-physician relationship. The physician is able to serve his patient with skill and understanding, since he is free to devote the time he feels necessary to the individual patient. He is free to prescribe the therapy that his knowledge and experience indicate as appropriate. However, as a human being, the doctor is concerned about his own economic welfare. Therefore, of necessity, the patient's ability to pay is a factor in their relationship. The concern of a patient over his projected bill is obvious to the physician. It is important that the physician relieve this anxiety in order that he and the patient may work together on the primary problem, the patient's illness. The time consumed in relieving this anxiety could better be devoted to relief of the illness. His prescription for medication or therapy may have to be bounded by how much the patient can pay, not necessarily by what would be most beneficial.

Another form of service is found in the prepaid group practice medical plan. In this type of plan, integrated groups of specialists are brought together under one roof. Potential patients subscribe to the services of these doctors for a fixed fee, usually payable monthly. The patient has the right to medical care when he needs it, according to the terms of the contract. Potentially, the services of every physician in the organization are available to him. The prepaid factor of this plan helps to relieve the patient's anxiety regarding finances. The services of this type of plan are limited to those living within the area where a major facility is located.

The member patient's choice of a physician is limited to members of the organization. It is frequently further limited by organizational policies or clerical procedures. For example, an emergency request for Doctor A is answered by the switchboard operator with, "So sorry, only Doctor Z is on call tonight." A call at the office may provide the information that Doctor B is no longer with the organization and Doctor C has no available appointments, but Doctor Y has a cancellation. As the patient passes from one to another on this medical assembly line, he begins to feel more like a part than a person. The recipient of conveyor belt care gets lost in the shuffle of charts and routines. He is a patient that passes through, not a person for whom the physician cares, for whom he works, and to whom he devotes the summary of his years of experience to help. There is little opportunity for the patient to feel secure through his relationship with the physician.

As an employee, the physician is more responsible to the organization than to the patient. His practice is regimented rather than self-regulated. The unique professional challenge of medicine is gone. The physician's identity is lost. He is a man with a job to do.

Compulsory government controlled health insurance and socialized medicine have been suggestions made by some. Cost of care to the individual would seemingly be minimized, but would, of necessity, be reflected in much higher taxes to all. The American system of free enterprise would be corrupted by such a plan involving the sacrifice of incentive, initiative, and individuality. Without these essential elements, the profession of medicine, as the healthiest nation in the world knows it today, would cease to exist.

Blue Shield, a nonprofit organization, conceived and controlled by the medical profession, offers still another plan in the form of voluntary health insurance. In Blue Shield, a subscriber insures medical treatment in advance by depositing small premiums

at regular intervals. When in need of medical treatment, he consults a physician of his own choice, who is paid entirely, or in part, directly from the Blue Shield fund. Relieved of financial stress, a patient will not put off seeing his doctor at the first symptom of illness. This will enable the physician to work more effectively with him. Secure in the knowledge that medical care is available when he needs it, and assured that it is not beyond his means, the stress of illness is not increased by anxiety over post-emergency costs. A Blue Shield subscriber knows that he himself may choose the physician in whom he has confidence. Thus the personal relationship so necessary to healing is on a stronger basis because the physician he goes to see as a Blue Shield member is *his* doctor.

The physician who uses Blue Shield service develops and regulates his own practice. Initiative is an asset, not a liability. Personal identity is not lost. Professional integrity is maintained at his own standards. As in all private practice, the physician's primary responsibility is to the patient he serves. Since the practice is his own, he is able to have the type of relationship with his patient that he desires. A Blue Shield subscriber is *his* patient and the personal satisfaction of purposeful practice is his. When a Blue Shield patient comes to the physician, the fee communication barrier is lowered because the Blue Shield schedule of fees provides the basis for opening discussion.

Essentially, Blue Shield serves as a reservoir of funds to which the patient deposits regularly and from which the physician withdraws when he gives treatment. It is operated solely for the benefit of the patient and the physician. No profit-seeking third party is involved. It is not without flaws, but it is flexible. Changes, as needed, can be made through the physicians who direct its operation.

This student has compared Blue Shield with other forms of practice on the basis of his own standards of service. He has found no plan without merit, and of course, no plan that is perfect.

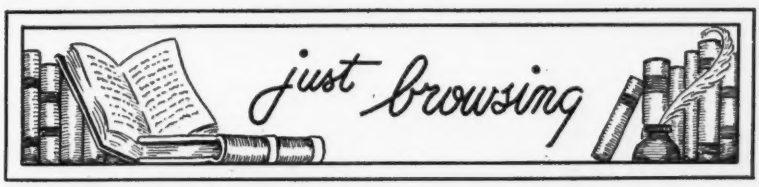
He has found in Blue Shield a plan that preserves the vital personal relationship between patient and physician, provides a professionally led program of competent care, protects the patient from the financial catastrophe of illness and injury, promotes physician incentive and initiative, and presupposes progress by a flexible form of operation.

This student of medicine looks forward to a professional career in which his personal satisfaction will come from his professional ability to help others. Blue Shield fits into such a professional career, helping him meet the challenge of providing medical care for his fellow man.

This medical student today is looking forward to becoming a Blue Shield physician of the future.

We will endure . . . if we accept our obligation to maintain stability in our world by conserving the traditions and institutions of the past and encouraging the orderly forces of progress. Those of us who assume that the capitalist society can and must endure rally around these standards. We believe in property but know it can flourish only when innovation has free rein and new enterprises (with the personal rewards that must go with them) can emerge. We are devoted to the idea of equality . . . of opportunity to rise. We are committed to the idea of man's rationality and his ability to make free choices for his own betterment and that of society.

—*Louis M. Hacker*



Small-pox is a disease which has been known in India and China from the earliest days of recorded history, but it was not until about the year 910 that a definite account of the disease was written by Rhazes (his full name was Abú Becr Mohammed Ibn Zacaríyá Ar-Rází), a physician in Baghdad.

"We will now begin therefore by mentioning the efficient cause of this distemper, and why hardly anyone escapes it; . . .

"I say then that every man, from the time of his birth till he arrives at old age, is continually tending to dryness; and for this reason the blood of children and infants is much moister than the blood of young men, and still more so than that of old men. And besides this it is much hotter; as Galen testifies in his Commentary on the 'Aphorisms'. . . For this reason the blood of infants and children may be compared to must, in which the coction leading to perfect ripeness has not yet begun, nor the movement towards fermentation taken place; the blood of young men may be compared to must, which has already fermented and made a hissing noise, and has thrown out abundant vapours and its superfluous parts, like wine which is now still and quiet and arrived at its full strength; and as to the blood of old men, it may be compared to wine which has now lost its strength and is beginning to grow vapid and sour.

"Now the Small-Pox arises when the blood putrefies and ferments, so that the superfluous vapours are thrown out of it, and it is changed from the blood of infants, which is like must, into the blood of young men, which is like wine perfectly ripened; and the Small-Pox itself may be compared to the fermentation and the hissing noise which take place in must at that time. And this is the reason why children, especially males, rarely escape being seized with this disease, because it is impossible to prevent the blood's changing from this state into its second state, just as it is impossible to prevent must (whose nature it is to make a hissing noise and to ferment,) from changing into the state which happens to it after its making a hissing noise and its fermentation. And the temperament of an infant or child is seldom such that it is possible for its blood to be changed from the first state into the second by little and little, and orderly, and slowly, so that this fermentation and hissing noise should not show itself in the blood; for a temperament, to change thus gradually, should be cold and dry; whereas that of children is just the contrary, as is also their diet, seeing that the food of infants consists of milk; and as for children, although their food does not consist of milk, yet it is nearer to it than is that of other ages; there is also a greater mixture in their food, and more movement after it; for which reason it is seldom that a child escapes this disease. . . .

"As to young men, whereas their blood is already passed into the second state, its maturation is established, and the superfluous particles of moisture which necessarily cause putrefaction are now exhaled; hence it follows that this disease only happens to a few individuals among them, that is, to those whose vascular system abounds with too much moisture, or is corrupt in quality with a violent inflammation; or who in their childhood have had the Chicken-Pox, whereby the transition of the blood from the first into the second state has not been perfected. . . .

"And as for old men, the Small-Pox seldom happens to them except in pestilential, putrid, and malignant constitutions of the air, in which this disease is chiefly prevalent. For a putrid air, which has an undue proportion of heat and moisture, and also an inflammed air, promotes the eruption of the disease, by converting the spirit in the two ventricles of the heart to its own temperament, and then by means of the heart converting the whole of the blood in the arteries into a state of corruption like itself. . . ."

It sounds a little complicated, but isn't it always complicated when we try to explain something which we ourselves don't understand?—*O.R.C.*

PRESIDENT'S PAGE

DEAR DOCTOR:

God, in His infinite wisdom, chose to take John Porter. His place is empty.

Gone is a devoted husband and father. Stilled is his unbounded curiosity, his interest in everything about him. Silent is his original and picturesque eloquence.

Gone is John Porter the physician. Departed is his uncompromising idealism, his impassioned crusade for ethics and high standards of care, his devotion to his profession.

Gone is John Porter president, at the beginning of a brilliantly planned year.

Gone—but no. In the memory of those who loved him, he is not away. In the lives of those to whom his life was an inspiration, he has an immortality on earth. His ideals continue to live among us. Our president is with us yet.

God, in His infinite wisdom, has given us John Porter's example to have with us always and forever.

It is for us, the living, to carry on with John Porter's plans for the Kansas Medical Society. The committees will stand as appointed with, possibly, an additional member being added to some. We must persevere that John Porter's example be not in vain. To this purpose I dedicate myself, so that, with your help, Kansas medicine may progress. May we, therefore, find increased satisfaction in the exercising of our profession.

Sincerely, in the Practice of the Art,

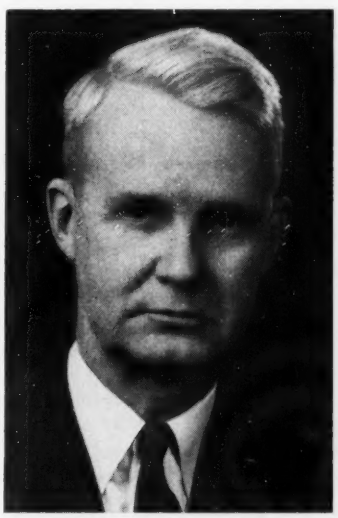
Conrad M. Barnes, M.D.

CONRAD M. BARNES, M.D.

Time May Have Been Cheated In an Untimely End

Today, over the state, they are saying that John McGill Porter of Concordia, president of the state medical society, died too young. But I wonder.

He died too young for his family and friends who loved him, and for his patients who leaned on him heavily. He died too young from the point of the medical society which had counted on the guidance of his intellect and his good judgment. But whether he died too young from his own point of view I seriously doubt.



JOHN M. PORTER, M.D.

Who is to say what is "too young"? John, with his intellectual honesty, would be the first to admit that science knows little or nothing about the twin mysteries of time and space. Today we take wings in an airplane and we fly into the future, or we fly in another direction and hold back the dawn. What is a day or a year? And what are 56 years—which was the span of John's life?

I only know that within those 56 years he did an incredible amount of living, and he did it zestfully. He accomplished more in the 26 years of his medical practice than most men accomplish in 50 years. He learned about history, mythology, philosophy, natural science. For he read avidly in non-professional literature. He participated in two wars, he had unusual experience in travel and in foreign study. For his hungry mind was always reaching out for experience. He crowded his leisure time with enjoyment of his family, his reading, his hobbies. He lived to know and love his grandchildren. And he realized some of his dearest ambitions.

What is old and what is young? I think John Porter lived a very long life indeed. And he did it without ever walking the tragic down-hill road, without ever knowing those last years spent in the shadow of personal loss and loneliness, without ever knowing the diminishing vitality, the faltering step, the dimming eye. In a way John cheated the thing we call time. For he lived at least 90 years, yet he died a vigorous man in the prime of life.—Marion Ellet in *Concordia Blade-Empire.*

EDITORIAL COMMENT

CULTS

Editor's Note: Dr. John M. Porter wrote this thoughtful, personal opinion on the subject of cults after attending a meeting in the interest of the Kansas Medical Society, only a few days over a month before his death. Believing the membership has an especial interest in this last formal extended writing by the president, it is printed here just as Doctor Porter wrote it.

July 23, 1955

Oliver Ebel, Executive Secretary,
Kansas Medical Society,
315 West Fourth Street,
Topeka, Kansas.

Dear Oliver:

This has been an exhausting trip but perhaps I can set down for you some of my ideas regarding cults and the practice of medicine. These are entirely unsolicited and like other forms of free advice may be worth exactly what they cost. However, writing it out will help to clarify my own ideas and if you can find any occasion to use the result, I hope you will do so.

These are individual opinions but I am sure they are shared by many other members of the Kansas Medical Society.

The practice of medicine is, of course, an art and consists and always will consist of the ministration of one individual to another. From the dawn of history until the relatively recent past this ministration has been interwoven and confused with the magical or with the priestly function. Personal magnetism, suggestion and a mutual feeling of confidence played a large part in "cures" or the alleviation of symptoms. The laying on of hands was often essential. Conceivably, there could be as many plans for treatment as there were "medicine men."

In the past 50 years all of this has become changed due to the increasing reliance of medicine on exact science. A patient's confidence is still a highly desirable thing but a fighting, struggling child will respond to diphtheria antitoxin and an inflamed appendix can be cured by good surgery even though its possessor believes only in divine healing. The comatose diabetic may never see the resident who saves his life and the asthmatic patient may be too sick to know who treats him. Modern surgery has become such a matter of team work that it would be difficult or impossible to divide the credit for success between the anesthesiologist, the various members of the surgical team, the pathologist who examines a frozen section and even the research worker whose animal surgery made the operation possible.

What part does the cultist play in all this? My answer would be "none." In functional disorders, incurable disease and in the all-important matter of reassurance in minor affairs he might seem to have a place but what assurance is there that the comforting, simple and readily available laying on of hands may not take place in cases of early malignancy, dangerous psychoses or these border line cases that tax the ingenuity of our best diagnosticians? Recognition of many diseases depends entirely on a high index of suspicion coupled with a scrupulous honesty that refuses to settle for the temporary relief of "manipulation." A few weeks frittered away in unscientific handling can very easily "fritter away" a life.

To understand these cults and their widespread acceptance it is necessary to examine the schooling of the physician and the universal reaction of the uneducated to scientific training. The student, immature and over-anxious, wants contact with the patient, he wants dramatic cures, he wants immediate rewards in terms of money and prestige. The long years in the laboratories, the cautious approach to human life and welfare, the long supervision of internship and residency are irksome and apparently illogical to many minds. When someone offers a short cut to the distant goal, a "secret" way to avoid the tiresome training and a quick answer to obscure questions, many simple-minded souls and some others more ambitious than honest grasp at these easy solutions. In this way the alchemists bypassed the harsh discipline of chemistry that finally learned how to transmute metals.

The astrologer tried the same plan with astronomy and managed to avoid a lot of mathematical drudgery. Medicine has been repeatedly attacked by cults with wonderful names and strange theories. This will probably always be so but this is no reason to capitulate. Truth has a way of being perpetuated that no pat theory however glibly explained or however apparently plausible can imitate. Science, of course, is merely a collection of truths assembled into working order.

It has been argued that homeopathy was absorbed by medicine and that other and newer types of capsule wisdom should be similarly absorbed. Homeopathy, however, never left the discipline of medicine and its students studied as long and as hard as any other students of medicine. The absurd con-

clusion that "like cures like" withered on the vine and was forgotten as homeopathy was forgotten, it was never absorbed into medicine.

Scientific medicine did learn two things from homeopathy, one that massive doses of drugs which had become fashionable were unnecessary and could be dangerous and second, that experimental pharmacology was a better measure of what drugs would do than some opinion based on the ideas of Galen. It is strange that such an odd and irrational cult should justify its brief existence by these two valuable concepts.

If more recent cults could claim any such virtues their other absurd claims could be more easily overlooked. No iota of progress can be honestly claimed by osteopathy and chiropractic in the many years of their existence. Even with their attention focused on the spinal column they failed to recognize the importance of injury to the intervertebral disc and it took a classmate of mine to describe these lesions for the first time.

Going from theory to practice we face the fact that cultists of various sorts are entrenched in most states of the union, practicing medicine and surgery either legally or as bootleggers. Often this knowledge was acquired in a pathetic imitation of a medical school but was always combined with instruction in the magic laying on of hands which even their teachers admit has no scientific basis. Graduate work for these practitioners is limited or non-existent and much of their "education" is through the lay press or from the advice of salesmen.

Several fallacies have recently arisen. One is that in attacking the osteopath's attempts to practice medicine and surgery we are ignoring more dangerous and less educated cults. This means to me embracing professionally a quack who is made up in good imitation of a physician and one whom many people think of only as another "variety" of doctor, in order to condemn more thoroughly the uneducated and untrained chiropractor whom none but the most ignorant would confuse with a physician. To my way of thinking the wolf in the best imitation of sheep-skin is the most dangerous.

Another mistake is to assume that if educational opportunities are equal the cultist must be as well qualified as a physician. This can never be true because the cultist's thinking is always biased by the mistaken, unscientific assumption of his cult. Whenever the going gets rough—tricky surgery, newer therapeutics, difficult diagnosis—the cultist always has an easy way out. If the education were equal the school would be a medical school, although probably class B or C.

Claims have been made that osteopaths can hold down locations in border line locations that would be unattractive to a well-trained physician. This may be true but supplies the unfortunate sick of those communities with a sub-standard sort of medical care. In many instances the osteopath able to enter practice earlier because of shorter and less expensive training becomes entrenched in a town which could otherwise attract a physician. I know of any number of communities in Kansas which could support a physician if all their medical needs were channeled through his office but when the older established practitioner skims off the cream what is left is insufficient to keep a doctor happy and busy or even alive.

I have no quarrel with the cultist who follows his beliefs whether they are Christian Science or naturopathy. The trouble comes when he wants to practice medicine without the training. The answer in my opinion is to allow only graduates of medical schools to practice medicine. We all know of respected physicians who tried osteopathy only to find it inadequate and who then studied medicine and became true physicians. I would recommend this to any graduate of a cultist school who feels he wants to do more than he is trained or licensed to do.

Very truly yours,

/s/ John

J. M. PORTER, M.D.
President

JMP:br

PORTER MEMORIAL

A letter to the membership of the Kansas Medical Society has announced the creation of the John M. Porter Memorial Student Loan Fund as a project of the Committee on Endowment.

Money from the fund will be distributed in the form of loans to junior and senior students at the University of Kansas School of Medicine. The continuing use of the fund will serve as a memorial to Dr. John M. Porter, whose death last month during his term of office as president of the Society has deprived us of the guidance of a physician known for his idealism, integrity, and unbounded interest in the future of medicine.

Contributions may be sent to the Society office with checks made payable to Porter Memorial. Those who wish to claim tax exemption for their gifts to the fund may make checks payable to American Medical Education Foundation with an accompanying note stating that the amount is intended for the John M. Porter Memorial Student Loan Fund at the University of Kansas School of Medicine.

ACH

(you probably know every answer!)

Q. Which is today's most widely prescribed broad-spectrum antibiotic?

A. ACHROMYCIN — it's first by many thousands of prescriptions.

Q. What are some of the advantages of ACHROMYCIN?

A. Wide spectrum of effectiveness.
Rapid diffusion and penetration.
Negligible side effects.

Q. Exactly how broad is the spectrum of ACHROMYCIN?

A. It has proved effective against a wide variety of infections, caused by Gram-positive and Gram-negative bacteria, rickettsia, and certain viruses and protozoa.

Q. In what way are ACHROMYCIN Capsules advantageous?

A. For rapid and complete absorption they are dry-filled, sealed capsules (a Lederle exclusive!) No oils, no paste...tamperproof.

Q. Who makes ACHROMYCIN?

A. It is produced — every gram — under rigid quality control in Lederle's own laboratories and is available only under the Lederle label.

CHROMYCIN*

Hydrochloride
Tetracycline HCl Lederle

Lederle

LEDERLE LABORATORIES DIVISION AMERICAN Cyanamid COMPANY PEARL RIVER, NEW YORK

*REG. U.S. PAT. OFF.

Our New President

Dr. Conrad M. Barnes, Seneca, who was elected first vice-president of the Kansas Medical Society at its meeting in May, took office as president of the Society on September 5, after the death of Dr. John M. Porter. His first official act was endorsement of a project of the Committee on Endowment creating the John M. Porter Memorial Student Loan Fund.

No introduction of Dr. Barnes is necessary, but a review of his accomplishments and interests will serve to record biographical facts that may not be

Conrad M. Barnes, M.D.

generally known to the membership. He was graduated from the University of Kansas School of Medicine in 1936. Since then his services as a physician have been in the field of general practice, which he construes as meaning treatment of the whole person, not the complaint. In his home community he is regarded as a counselor, philosopher, and friend as well as a physician.

About ten years ago, when the Society established a Committee on Rural Health, Dr. Barnes was chosen as its first chairman. At the invitation of the Council on Rural Health of the American Medical Association, he attended a meeting at which the National Council on Rural Health was organized, and since that time he has been generous with his abilities and energies in that field. He was one of the speakers at the national council's meeting in 1954 and later presented his address over the radio on the National Farm and Home Hour.

The Blue Shield plan has benefited from Dr. Barnes' interest, and he served Kansas Physicians' Service as its second president, remaining in that office for a year and a half. He is also active in the Kansas Chapter of the American Academy of General Practice, in the preceptorship program of the University of Kansas School of Medicine, and in the medical school itself. In addition he gives freely of himself to his county medical society and was in part responsible for the construction of the Seneca Hospital.

Dr. Barnes' personal interests are as varied as his services to medicine. As a lover of fine music he has assembled a record library that is outstanding. He possesses a voice of pleasing quality and enjoys himself thoroughly when harmonizing with a quartet or larger group. He can describe the art of glass blowing, especially as it concerns his collection of rare bottles of all types, interesting because of their history or design.

Members of the Society are confident they will be guided wisely and well during Dr. Barnes' administration. As we wish him well, we pledge to him our wholehearted cooperation and support.

Obstetrical Society to Meet

A meeting of the Kansas State Obstetrical Society will be held in Kansas City on Tuesday evening, November 8, a date which will be convenient to many physicians practicing obstetrics and gynecology since a refresher course on that subject will be presented at the University of Kansas Medical Center on November 7, 8, and 9.

The exact time and place of the meeting will be announced to the membership later, but it is known now that Dr. Lawrence Randall, of the Mayo Clinic, Rochester, will be guest speaker. Members are invited to bring their wives to the dinner session.

Any member of the Kansas Medical Society who is interested in obstetrics and gynecology is eligible for membership in the specialty group. A check for $5.00, payable to the Kansas State Obstetrical Society, covers membership fee. It should be mailed to Dr. Edward Crowley, 435 North Hillside, Wichita, secretary-treasurer.

Surgeons Elect Officers

Dr. Thomas P. Butcher, Emporia, was named president of the scientific section of the Kansas Chapter of the American College of Surgeons at a meeting held in Emporia on September 11. Dr. John Grove of Newton was named president-elect, and Dr. Orville R. Clark, Topeka, was elected secretary-treasurer.

Advertisers make possible the publication of this Journal. They will appreciate the patronage of Journal readers.

Clinicopathological Conference

CASE PRESENTATION

Dr. Youmans (neurosurgery resident): This 71-year-old colored man was admitted to KUMC on September 21, 1954, and expired on September 28, 1954. The chief complaint was shortness of breath for two months.

The patient stated that he was well until July, 1953, when he realized that because of weakness of the right arm he could not raise it above a horizontal level. Approximately one month later he developed the habit of dragging his left foot while he walked. By January, 1954, the patient required a cane to walk and eventually used a crutch. Two months prior to admission he became unable to walk because of his knees buckling. He had noted that, intermittently, his muscles "jumped" since January, 1954.

The patient had a past history of the usual childhood diseases without sequelae. He had gonorrhea at age 18 and smallpox at age 25. At about this same age he fell three stories from a window to the sidewalk without apparent injury. At age 48 he was struck on the head by a brick which fell one and one-half stories, causing a "blood clot on the brain" and double pneumonia to develop. Seven years ago he became aware that water made his skin feel like hot grease and had used water as rarely as possible, always following it with baby oil.

Approximately two months prior to admission the patient developed shortness of breath, orthopnea, paroxysmal nocturnal dyspnea, and a hacking cough productive of small amounts of mucoid sputum. He had slept in a sitting position for two months. Moderate swelling in the ankles had been noted for this same period. For the past month he had indigestion and mild constipation. His usual weight was 160 pounds. He had had nocturia four times nightly during the past month. He smoked 50 cigars a week and was a moderate drinker.

The family history was non-contributory except to show that the parents and children were long-lived, the mother dying at 84 and the father at 90 years.

On physical examination, the patient was a well developed, well nourished Negro man who appeared moderately dyspneic. Blood pressure was 120/70, pulse 84 and regular, respirations 28. The pupils were round, equal, and responded to light and accommodation. Accessory teeth were present extending out into the floor of the mouth. There was no cervical, axillary, or inguinal lymphadenopathy.

Decreased fremitus and breath sounds with dullness to percussion were present bilaterally in the bases with moist rales in the upper lung areas. Point of maximum impulse was not palpated. The area of cardiac dullness was moderately enlarged to percussion. There were no thrills or murmurs.

The liver, kidneys, and spleen were not palpable. No abdominal tenderness was elicited.

Three plus pitting edema of the ankles was present. Deep tendon reflexes were absent on the right upper extremity. Babinsky and Hoffman signs were absent. No sensory changes were elicited. There was marked weakness of all voluntary muscles with complete paralysis of the right upper extremity. Marked atrophy of the small muscles of the hand was present on the right. Fasciculation of the left facial muscles was seen. There was some weakness in the sternocleidomastoids.

Laboratory Examination: Urinalysis: reaction alkaline, specific gravity 1.020, albumin negative, sugar negative, microscopic 5 to 10 pus cells/high power field, occasional hyaline casts. Red blood count was 5,000,000 with 14.5 grams hemoglobin; white blood count 5,040, of which 62 per cent were polys., 32 lymphocytes, 4 eosinophiles, and 2 monocytes. VDRL was reactive with 16 Kolmer units; nonprotein nitrogen 30, creatine 1.2, sugar 80 mgm./100 ml., sodium 134 mEq./L., potassium 6.2 mEq., CO_2 27 mEq., chlorides 98 mEq./L. Twenty-four hour urine volume was 600 ml.

There is some confusion on the protocol regarding creatinine levels. In the protocol it is marked creatine, and both of these should be marked creatinine. The technique is essentially this: The urine is checked first for creatinine and the value obtained on this was preformed creatinine which was 75. There seems to be some discrepancy on normal values. Values for this laboratory range from 700 to 2000 mgms. per 100 ml. Then the urine is treated with hydrochloric acid and leaded straps, and in this process all of the creatine present is converted to creatinine. It is re-run then as the total amount as recorded here, again as creatinine, not creatine. The total was 152, and the difference between the two values then will be the amount of creatine present in the urine which was 77. In this laboratory the normal range is from 0 to 50 mgms. per 100 ml. Sedimentation rate 18 mm. fall/hr. I^{131} uptake 7 per cent. Absence of denervation fibrillation potentials was noted in the electromyogram. Fasciculation potentials were present.

After the patient was admitted to the hospital, initial therapy consisting of low sodium diet, digitoxin, and aminophylline brought prompt response with loss

Edited by Glen R. Shepherd, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, pathology, physical medicine, radiology, and surgery of the University of Kansas Medical Center as well as by the third and fourth year classes of medical students.

of the edema and clearing of the lungs. Other therapy consisted of vitamin E, folic acid, and multi-vitamin capsules. Although the rales cleared in the lungs, poor aeration was recognized, and the patient was started on procaine penicillin. He was clinically much improved with correction of the congestive failure, but he continued to be extremely weak and bedfast. On the afternoon of September 27, 1954, the patient developed respiratory distress with poor diaphragmatic excursions. This became more marked the following morning, but he still seemed to be aerating adequately and without cyanosis. Respirations became progressively more shallow until mid-afternoon when he became cold and began sweating profusely. Blood pressure was still in its usual range of approximately 120/70, but it began to fall about two hours later. As the blood pressure began to fall, the patient had a sudden onset of respiratory distress with cardiac arrest. Pounding on the chest brought about resumption of the cardiac rate. The patient was given artificial respiration for a few moments before the heart again stopped. He was placed in a respirator, given nasal oxygen and 1 cc. of 1:2000 prostigmine intramuscularly. The patient died one-half hour later.

Dr. Delp (chairman): Any questions of Dr. Youmans?

Question: What was the mental status of the patient?

Dr. Fuente (neurology resident): He was quite alert. The mental status was considered normal.

Question: Tell us more about this seven years of queer skin sensations.

Answer: That's all the information we have. He found that water in bathing or washing made his skin burn. This bothered him to such an extent that he used it only when he felt he really had to, and he always followed it immediately with talcum powder or baby oil.

Question: Was that all his skin or just his right hand or what?

Answer: It seemed to be generalized over his entire body.

Question: Was this man really a well developed well nourished individual?

Answer: That is why I prefaced my remarks by saying that from gross appearance, if you walked in and looked at him, you would find nothing grossly wrong. He was not an ill developed, malnourished, or an emaciated individual.

Dr. Delp: His weight in March was 158 pounds.

Question: I was wondering about reflexes in the extremities. They were not mentioned in the protocol. How about the extremities and the left arm, or abnormal reflexes?

Dr. Fuente: With the exception of the deep reflexes, there were no reflexes present anywhere.

Question: Was the spinal fluid negative?

Answer: Yes. The spinal fluid washings were negative, the colloidal gold was negative, 2 white blood count, 458 red blood count, total protein 116.

Question: What did his tongue look like?

Answer: His tongue was grossly normal.

Question: What about his eye grounds?

Dr. Fuente: They were normal.

Question: During the hospital course, did the patient complain of pain in the legs or chest?

Dr. Fuente: The patient had no pain.

Question: As concerns reflexes, did you check his abdominal reflexes?

Dr. Fuente: The abdominal reflexes were absent.

Question: Had the patient ever been treated for syphilis?

Dr. Youmans: As far as the chart is concerned, no. He did specify that he had gonorrhea at age 18 and that it was treated.

Dr. Delp: I think that the question had been asked of the patient, and he stated that he had not been treated for syphilis.

Question: About the 450 red cells in the spinal fluid. That wasn't an error in the tap?

Answer: I think you will have to draw your own conclusions as to that.

Question: How long had this patient been seen in the outpatient clinic?

Answer: Since March, 1954.

Question: What did he complain of when he came to the outpatient clinic then?

Dr. Delp: Weakness of the right arm. "Loss of use of the right arm, left leg feels like it locks when walking. Itching over the body following soap." The patient was 68½ inches in height, weighed 158¾ pounds. His temperature was 98.2° F. when he came into the hospital. He had a 52-year-old wife.

Mr. Albert P. Simpson (fourth year student)*: The electrocardiogram standardization is normal, the rate about 86, with regular sinus rhythm. The P waves are normal appearing. PR interval is about 0.18 second, which is the upper limit of normal. QRS interval is not abnormally wide nor tall. My diagnosis would be digitalis effect and left ventricular strain.

Dr. Delp: Is there anything in the electrocardiogram, Simpson, that might portend such a death as described in this patient?

Simpson: Not to me, sir.

Dr. Delp: Dr. Lin, do you have any comments about the electrocardiogram?

Dr. Lin (cardiologist): No, sir.

Dr. Delp: Dr. Tice, do you have any comment about the x-ray films?

Dr. Tice (radiologist): The cervical spine could be a little abnormal. I think the sixth cervical verte-

* Received M.D. degree since this conference, in June, 1955.

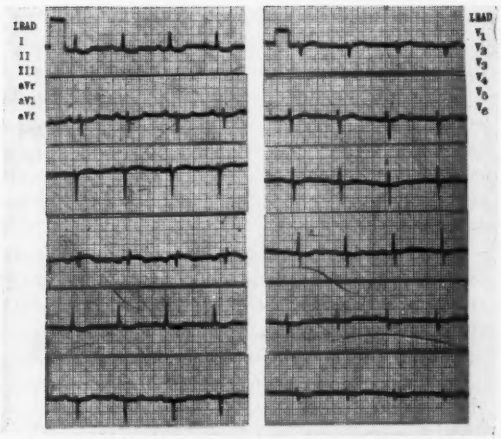


Figure 1. Electrocardiogram taken September 23. Interpretation in text.

bra is depressed, and I think it projects back toward the cervical canal, but I can't be sure. It might be due to osteoarthritis with a proliferation of bone back toward the canal. The mediastinum is definitely widened.

Dr. Delp: As you noted on the protocol, there was an electromyographic tracing made on this patient. I want to call on Dr. Rose to tell us about this tracing in general. We just want the findings, Dr. Rose, not your diagnosis.

Dr. Rose (physical medicine): The resting record in this case was taken with the electrode inserted into the adductor pollicus brevis. This muscle was selected because fasciculations were noted in that muscle. When the patient is at rest the record should be electrically silent, with no potentials noted above the normal level of electrical interference. This was the case in this patient.

There were no spontaneous appearing electrical potentials with the exception of occasional fasciculation potentials. These were manifest by single or occasional runs of two or three reasonably normal appearing motor unit potentials, again appearing in the resting record. When attempting to contract the muscle, a normal height of motor unit potentials

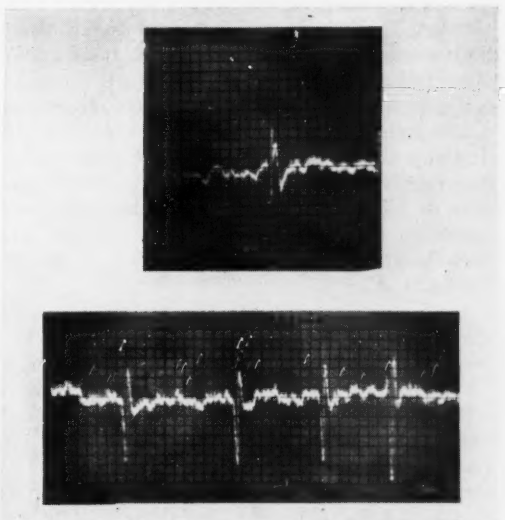


Figure 3. Electromyogram. The upper tracing shows a triphasic fasciculation potential in the resting record. The lower tracing shows the electrical response on maximal voluntary effort which, although indicative of an intact lower motor neuron, demonstrates potentials of reduced frequency, voltage and duration from normal.

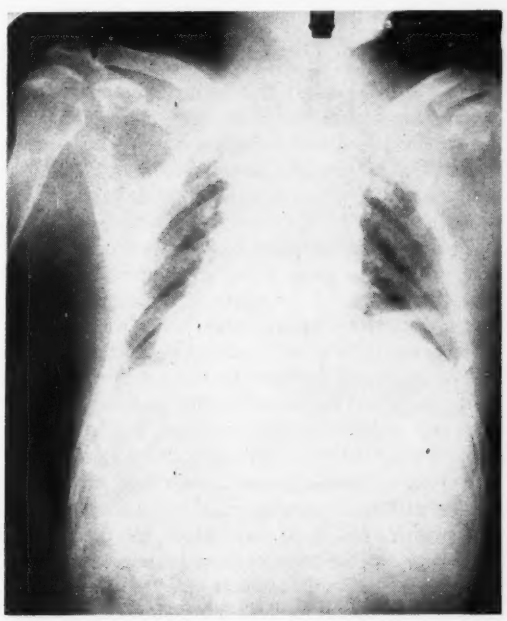


Figure 2. X-ray of chest. Interpretation in text.

should be seen, characterized by a certain given height of voltage and a certain duration of the potential in the same sense as in a normal electrocardiogram. In this patient, the potentials were briefer than normal in duration, of smaller amplitude than normal, and they were of diminished frequency.

Dr. Delp: Thank you, Dr. Rose. If there are any uninitiated here we will ask you to elaborate later concerning this procedure. I suppose that the clinical diagnosis in this case might be perfectly apparent to everybody, especially those people in the third row. I don't think it was entirely apparent to the staff, at least it doesn't seem to be evident from the diagnostic sheet record. It does seem rather strange that a man who had had such vigorous health throughout life should have deteriorated so rapidly after coming into

the hospital. For that reason I would like to have explained exactly why this patient died.

DIFFERENTIAL DIAGNOSIS

Mr. Ralph H. Wood (fourth year student)*: Briefly reviewing a few of the more important features of this case, an aged Negro man entered the hospital with the complaint of shortness of breath and presented the classical symptoms and signs of congestive heart failure. Quite rapidly the congestive heart failure improved upon therapy with digitalis, aminophylline, and a low sodium diet. In spite of this, and even though his congestive failure was corrected, the respiratory distress became progressively worse. Aeration seemed to be exceedingly poor and there was limited movement of the diaphragmatic excursions. The last day, which was the eighth day of hospitalization, his respiratory distress became even more marked and he died.

The present illness was characterized by weakness of the right arm 14 months prior to death and a dragging of the left leg 13 months prior to death. The muscles "jumped" intermittently nine months prior to death. For the past two months he had been unable to walk because both knees buckled. The paresthesia of the skin for seven years is an interesting bit of past history.

On history alone the first and last thing I would think of would be amyotrophic lateral sclerosis. This is a classical history of amyotrophic lateral sclerosis. The significant physical findings are that he had no sensory loss and had complete areflexia (no reflexes at all, pathological or normal, except for the pupils), fasciculations of the left facial muscles, atrophy of the smaller muscles of the right hand, complete paralysis of the right upper extremity, and limited control of the other extremities.

The significant laboratory findings were a positive VDRL with a Kolmer of 16 units, a slightly above normal creatine excretion, an I^{131} uptake of 7 per cent which is below normal, an absence of denervation potentials on the myogram, and a negative spinal tap. I would interpret the spinal fluid red blood cells as due to a traumatic tap which also produced the apparent increase in proteins.

The differential diagnosis, I think, should concern the causes of muscle atrophy, which are many.

Syringomyelia might be thought of, but it usually produces a wasting of the muscles of the upper limb, often bilaterally, and spastic weakness of the lower limbs; fibrillations are rare. Always there is a segmental loss of pain and temperature and always there are sensory changes.

Syphilitic amyotrophies or syphilitic meningomyelitis might be considered by many. Usually this is accompanied by pain of considerable severity. We do have a history of paresthesia of seven years' duration. Usually the signs of pyramidal degeneration are lacking, and in this patient they were lacking. Usually the patient has pupillary abnormalities, but this patient had normal pupils. Usually there is evidence of syphilis in the cerebrospinal fluid. This patient had no such evidence of syphilis. I would be inclined to rule out these syphilitic diseases.

Spinal cord tumors should be thought of in these patients. Usually such tumors produce muscle atrophy in one or both upper extremities with a spastic paraplegia, which this patient certainly did not have. Always they produce a sensory loss. On spinal tests, there is a positive Quackenstedt test and a high spinal fluid protein, which I don't think this patient would have shown if red cells hadn't been there. Spinal tumors often produce nerve root pain in the arms, which this patient certainly did not have.

This patient could have had a herniated cervical disc, in which case there would have been pain in the arm and only unilateral motor reflex signs. This patient had motor reflex signs absent bilaterally all over the body with fasciculation in the face, which I think definitely rules out a herniated disc.

I would be inclined to rule out a cervical rib practically on the same basis as a herniated cervical disc as well as because of lack of x-ray evidence of a cervical rib.

A radiculitis or inflammation of the spinal nerve is another possibility. This is most common in the fifth cervical nerve, and we did have evidence of arthritis in the cervical x-rays, which could have contributed to this. There is usually a wasting and weakness of muscles which the fifth cervical nerve supplies. This patient had a wasting and weakness of many muscles. Usually such radiculitis is of acute onset. The patient had severe pain. But it is never progressive, whereas our patient became progressively worse.

This patient could have had peroneal muscle atrophy, which is a progressive weakness involving the feet and legs, later spreading to the arms. It never extends above the elbow. The cranial nerves are always normal. This patient had fasciculations of the left facial muscle. Typically there is a foot drop which this patient did not have. Typically it begins at an early age and is strongly hereditary. We have a history of this patient's parents and all his brothers and sisters living to an old age.

This patient conceivably could have had myasthenia gravis with a history of easy fatigue, but there would have been absence of atrophy and fasciculation. Such patients do respond to a test dose of prostigmine; granted this patient received prostigmine, it was probably too late for effectiveness. If it had been given

* Received M.D. degree since this conference, in June, 1955.

earlier, it probably would have increased the fasciculations as most commonly happens in amyotrophic lateral sclerosis.

The muscular dystrophies should be considered briefly. They are ruled out on the basis of the test done by Dr. Rose and on the basis of the excretion of creatine. There is never fasciculation in the muscular dystrophies.

This patient could have had thyrotoxic myopathy except that his I^{131} uptake is 7 per cent and he exhibits no signs of thyrotoxicosis.

Finally, we come to the nuclear amyotrophies or motor neuron diseases. Actually this is a difficult subject to discuss because everyone who has written on it has a different classification. However, this group of diseases or disease, depending on whom you read, is characterized by degenerative changes of the anterior horn cells of the spinal cord and/or the brain stem and pyramidal tract, degeneration due to an upper motor neuron degeneration. There is atrophy and weakness of the affected muscles. There are reflex changes which are variable; they may be spastic and they may be flaccid, depending on where the lesion is the worst. There is characteristically in this group of diseases an absence of sensory changes. Usually they occur in middle life, but they may occur as late as the 70s. There are many cases in the literature of men 70 years old having this group of diseases. They are progressive types of diseases. Usually the length of life is from two to five years after onset, although there are patients on record living as long as 20 years. Many different things are confused, and I will mention four large groups.

One group is progressive muscle atrophy, in which the lesion is essentially in the anterior horn cells of the spinal cord. There is no other motor neuron lesion, there is no lateral tract involvement. This disease offers the best prognosis, many of the patients living to be 20 years old. We know this patient did not have this disease because he did have involvement of the left face, which you would not have from just a cord involvement.

Next there is progressive bulbar paralysis where the degeneration of the anterior horn cells of just the medulla and pons occurs. Usually these patients die in respiratory failure. They have difficulty swallowing and in phonation. They have a typical atrophy of the muscles of the tongue, which, if you see, you will never forget. This patient obviously did not have this. At least he didn't have the tongue muscle atrophy.

Another group of these diseases is primary lateral sclerosis, which is due to an upper motor neuron lesion with secondary sclerosis and demyelinization of the lateral corticospinal tract. This patient obviously did not have this disease alone because he did have signs of lower motor neuron disease.

Finally, there is amyotrophic lateral sclerosis, which some people would call all of these diseases. They would put them all under the heading of amyotrophic lateral sclerosis, which is a lower motor neuron degeneration with pyramidal tract involvement and/or medullary involvement. Usually these patients die within two or three years, and the prognosis is usually the worst with this disease.

I am unable to demonstrate pyramidal tract involvement in this patient because of his complete areflexia, yet I still think the pathologist will show us this. So I do think this patient had primary amyotrophic lateral sclerosis, and I think he died in progressive respiratory failure. I cannot rule out syphilitic myopathy though I don't think the patient had this.

The secondary findings which I think the pathologist will show us will probably be a syphilitic aortitis. I hope he didn't have an aneurysm although he could have had. He will have had a pneumonitis due to the prolonged respiratory failure. I think there was also arteriosclerotic heart disease with hypertrophy.

Dr. Delp: Thank you. You know, Wood, you are really reassuring. Medicine is not as difficult as I thought it was.

CLINICAL DISCUSSION

Dr. Delp: Dr. Steegman, would you comment about this patient?

Dr. Steegman (neurologist): I would like to say a few words first about the semantics. I heard the words positive Quackenstedt and positive Babinski. I don't know what a positive and negative something is when you try to define it in terms. I think those signs should be recorded as present or absent. If you are speaking of the spinal fluid dynamic test, there should be some elaboration of what you mean by positive Quackenstedt, because I don't understand what you mean by positive or negative.

This man obviously had a motor disease without sensory change. He did have a peculiar skin complaint, but there was nothing typical about it and he could respond quite adequately to sensory tests. So we decided that his disease had no real sensory component. There were signs of lower motor neuron type of disease indicated by the progressive atrophy and muscle weakness with fasciculation.

Here again, I don't know what a fibrillation potential is. You do not get fibrillation of a muscle in lower motor neuron disease. You get fasciculation, because a single anterior horn cell supplies about 200 muscle fibers. Dr. Rose probably will clear up what he means by that. At least clinically you would get fasciculation of a muscle and not anything you would call fibrillation, the type of thing you see in heart muscle.

This disease was much more extensive, I think, than was indicated by this history. The patient had

marked involvement of the lower extremities with fasciculations in the muscles of the lower extremities. So I think he had a form of lower motor neuron disease of a chronic progressive character that would fit into some sort of progressive spinal muscle atrophy. I believe personally that progressive spinal muscle atrophy and amyotrophic lateral sclerosis are the same disease. I think in each instance you are dealing with anterior horn cell degeneration, and the clinical picture in each individual depends upon which portion of the neuro-axis is involved.

With involvement of the Betz cells of the cortex, which are the large pyramidal type cells, then the upper motor neuron signs are conspicuous. If there is involvement of the anterior horn cells in the spinal cord, then the lower motor neuron signs would be conspicuous. If it happens to occur in the lumbar cord, then the weakness and degeneration will be greater in the lower extremities. If it occurs in the cervical cord, which is the typical place in amyotrophic lateral sclerosis as described in text books in its classical form, then there is weakness and atrophy in the hand and upper extremities and upper motor neuron signs in the lower extremities.

But we see some cases in which the disease is confined to the spinal cord primarily except in its late stage, when it may ascend to the bulb. Then there are signs of bulbar paralysis. Occasionally it may begin in the bulbar region first. Then the bulbar paralysis will be the most conspicuous part of the picture. The involvement of the limb may occur terminally or perhaps not at all, although there may be some minimal changes around the face.

I personally think this man died of respiratory failure. These patients have more and more weakness of the muscles of the thoracic cage. Then anything which increases the effort by a small amount will throw them into uncontrolled anoxia, and they will die rather rapidly. I think that is the most logical explanation in this patient.

Dr. Delp: Dr. Rose, I notice that on the report you sent from your department the diagnosis is stated in this way, "This electromyograph is consistent with the clinical diagnosis of amyotrophic lateral sclerosis." In making this diagnosis, did you disregard others of the possibilities brought up here?

Dr. Rose: The primary point of distinction is based upon the absence of fibrillation potentials. Perhaps since Dr. Steegman raised the question, I may say a word about that.

This is just a matter of definition. From the electromyographic standpoint, we regard fibrillation as pertaining to the activity which we relate to single individual muscle fibrils. Fibrillations are seen in lower motor neuron lesions in which the lower motor neuron is functionally disconnected from the muscle fibrils. Under those circumstances, one sees characteristic potentials which are of low voltage and are much briefer in duration than the normal motor unit type of potential. They have a different appearance in the cathode ray tracing, and they have a totally different sound when one screens them through the loud speaker.

The resting muscle was tested by three samples in this individual. It was totally silent for fibrillation potentials. There were, however, numerous fasciculation potentials which did appear spontaneously and were regarded as being evidence of "irritation" at the anterior horn cell level. In other words, something stimulated the anterior horn cell. Therefore, we find the normal motor unit type of action, and this very frequently coincides with a clinical twitch of the muscle as well. It, therefore, corresponds to a functional and anatomically intact lower motor neuron; otherwise normal appearing motor unit potentials would not appear.

The absence of fibrillation potentials would indicate that at least in the area sampled there was not a lower motor neuron type of involvement. The presence of a fasciculation potential would indicate that it is not something which is concerned with functional activity, which is part of the normal movement pattern. And, finally, the appearance of the low voltage somewhat briefer in duration than normal motor unit potential would indicate that there is functional integrity of the upper motor neuron, but it is of poorer quality (I use the word in a rough sense) than we normally expect to find. Increasing the effort failed to intensify the interference pattern that was developed. Therefore, this man was using about as much effort in activating the lower motor neuron as he could.

Question: Does fasciculation occur only when the axon body is diseased but not completely knocked out? If it is completely knocked out then you won't get fasciculation?

Dr. Rose: Conceivably it is possible that there be an upper motor neuron lesion which is of an irritating type so that there are efferent and spontaneous discharges across the upper and lower motor neuron tract at the anterior horn cell level in which you would find the appearance of fasciculation potentials. However, I should also point out that you do see them in connection with poliomyelitis as well, so that apparently it may occur in anything which would stimulate the lower motor neuron. It is a stimulatory type of response, and the fibrillation potential has nothing to do with the lower motor neuron.

Dr. Delp: Has anyone else any suggestions?

Dr. Fuente: We knew that this man's vital capacity was dropping, and there was no adequate way to improve it. We discussed the possibility of putting him

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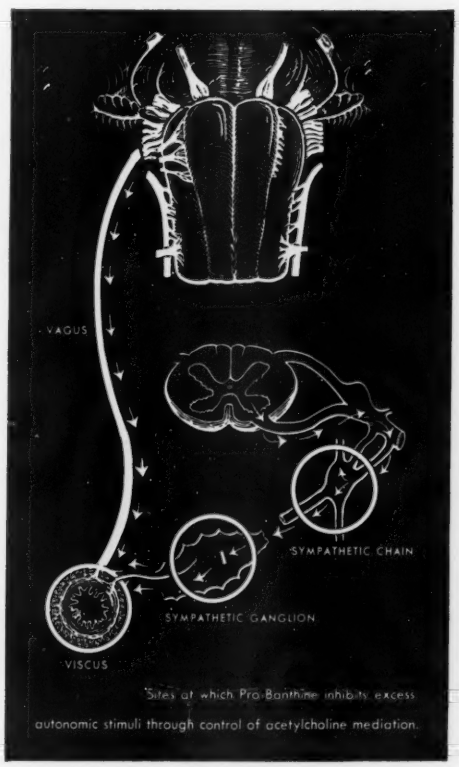

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1. Schwartz I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: Gastroenterology *25*:416 (Nov.) 1953.

2. Roback, R. A., and Beal, J. M.: Gastroenterology *25*:24 (Sept.) 1953.

SEARLE

in a respirator. He was able to count up to five or six before he became anoxic, which represents about 400 cc. He was extremely dyspneic, and we considered the possibility of having to put him in a respirator. We didn't want to do it considering the nature of his disease.

Dr. Delp: Dr. Lin?

Dr. Lin: Considering the heart tracing, I think I should consider rupture and the other possibility of coronary artery occlusion.

PATHOLOGY REPORT

Dr. Mantz (pathologist): It will be a distinct pleasure to confirm in large part the diagnosis made by the discussant. First, let me say that examination of the body and the first examination of all organs failed to reveal any significant features I could show you. At the completion of the autopsy we were in the dark as to what the nature of the lesion actually was. The pathologists were aware that this clinically was a disease involving the motor tracts, so we then directed our examination primarily to the central nervous system.

The brain was essentially normal, weighing 1240 grams. No localizing lesions were seen. The spinal cord likewise was free of abnormalities, although there was perhaps some slight flattening in the anteroposterior diameter.

Let us now proceed to the examination of the motor tracts throughout their length and perhaps be able to show some abnormalities.

A section of the motor cortex suggested that there had been some loss of the total number of neurons present. This was partially evident in the deeper areas.

A Nissl's stain of deeper brain layers, where Betz cells are normally seen, confirmed the observation, because we found only a few scattered remnants of the pyramidal elements which serve as the upper motor neuron of the motor system. Where Betz cells could be found in any significant numbers, they displayed definite evidence of degeneration, a rather marked swelling in the cytoplasm, and rounding and distortion of the outline. The nuclei seemed fairly normal.

Fat stains of the motor cortex disclosed an excessive amount of fat contained within the cytoplasm of the Betz cells.

A section from the base of the pons in the area of the crossing fibers of the pyramidal tract stained for fat showed a few scattered macrophages containing fat, presumably indicating myelin degeneration of the motor fibers in that area.

A section from the midportion of the medulla in the area of the ninth cranial nerve nucleus showed some loss or diminution of the normal number of neurons. One wonders if there might have been some palatal weakness in this case.

A section of the sixth nerve root showed some evidence of fatty changes. Now I am aware that this patient did not exhibit oculomotor weakness, and also that such changes are quite unusual in the disease we are attempting to demonstrate. However, the fatty changes would suggest that some degeneration occurred within the sixth nerve, at least in that area.

The major changes, as predicted, were present in the spinal cord. They were best seen in the lateral and ventral columns. A fat stain of the cord section showed extensive symmetrical deposition of free lipid in the region of the lateral columns.

In addition to lateral and ventral column involvement, there was extensive disease in the anterior horn, with diminution in the number of cells and rather extensive alteration in the individual nuclei. A fat stain emphasized the dearth of cells present.

As one might expect, the degenerative changes extended outward along the peripheral nerves. One to the branches of the brachial plexus showed rather marked fibrosis and collaginization of the epineurium,

Figure 4. Photomicrograph of section from posterolateral cervical spinal cord showing marked degeneration of myelin in the lateral columns, which can be recognized as black granules of liberated lipid.

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fibrosis within the nerve itself, and marked diffuse vacuolization along the nerve fibers.

Finally, we saw rather extreme degeneration and atrophy, marked by shrinkage of the fibers which appear excessively shrunken and wavy in outline. There was some interstitial fibrosis. Early interstitial invasion of fat was apparent, and a mild degree of concentration or proliferation of sarcolema nuclei was present.

These changes are characteristic of the classical picture of amyotrophic lateral sclerosis as the pathologist sees it at autopsy. I do not think that there can be reasonable doubt that the disease actually existed.

The remainder of the examination of the body was not too revealing. The lungs were studied carefully. They were relatively small, weighing only 450 grams, and hardly the lungs expected with a significant degree of cardiac failure with passive congestion. There was extensive adhesion present, marking perhaps the pneumonia that was described in the clinical history. The most dramatic changes, however, were found on microscopic examination.

Scattered throughout many sections of all lobes of both lungs were multiple remote and organizing pulmonary emboli. The results of this process, I believe, were reflected in the larger pulmonary arteries and in the heart. A section of the pulmonary artery, perhaps of the third or fourth order, showed a profound degree of sclerosis of the wall involving primarily the media and the intima with a rather extensive degree of atheromatous deposit.

The heart was not too remarkable in gross appearance. It was not heavy, weighing 350 grams. There was questionable hypertrophy of the left side, but, more significantly, the right heart appeared to be dilated and hypertrophied. The right ventricle measured seven millimeters in thickness. This is the picture that was described by Castleman and Bland and has since come to be known as the Castleman-Bland syndrome. It is a multiple embolization of the pulmonary vascular tree associated with cor pulmonale and certain classical clinical features.

We believe, in summary, that the basic cause of death in this patient was amyotrophic lateral sclerosis. We can contribute nothing to the understanding of the etiology of that process. Perhaps, however, the history does suggest the faint possibility of trauma. We do believe that the termination was related to the neurological disorder, but the fact that it was in some way mediated by this pulmonary embolization is too inviting a possibility to be completely overlooked.

Dr. Delp: Any questions of Dr. Mantz?

Wood: Did the aorta show any evidence of syphilis?

Dr. Mantz: The aorta was entirely free of such evidence. There was a minor degree of senile ectasia with atherosclerosis.

Wood: The mediastinum was not enlarged?

Dr. Mantz: The mediastinum was not significantly enlarged.

Question: Where did the emboli come from?

Dr. Mantz: That is the $64 question. We never found a source. The possibility that this was derived from the periprostatic veins cannot be overlooked. On the x-ray films, phleboliths were found in the pelvis. We were able to confirm their origin in the periprostatic areas. These may have been old thrombi which gave rise to the emboli, and the emboli were apparently of considerable age.

Nash: There was no coronary artery disease?

Dr. Mantz: Coronary involvement by atherosclerosis was minimal. There was no change in the muscle by histologic examination.

PATHOLOGICAL ANATOMICAL DIAGNOSIS

Primary

Generalized amyotrophic lateral sclerosis with degeneration of ganglion cells and loss of Betz cells in the motor cortex; degeneration of anterior and lateral corticospinal tracts and ventral horn cells (history of progressive muscular weakness beginning in the hands, particularly right, 14 months before death, and of the lower extremities nine months before death).

Atrophy of muscle groups including flexors and extensors of forearm and hands, right and left shoulder girdle, intercostal, flexors and extensors of lower legs and feet, advanced (history of neurological diagnosis of progressive spinal muscular atrophy five days before death and of clinical and electromyographic diagnosis of amyotrophic lateral sclerosis two days before death).

Multiple remote and recanalized emboli of pulmonary arteries, advanced, with remote and recent pulmonary infarcts, slight (history of increasing dyspnea and orthopnea, with paroxysmal nocturnal dyspnea, during the last weeks before death).

Pulmonary arteriosclerosis, moderate.

Hypertrophy of right ventricle of heart.

Atelectasis of the right lower lobe of the lung, advanced, and of all other lobes of the lungs, slight.

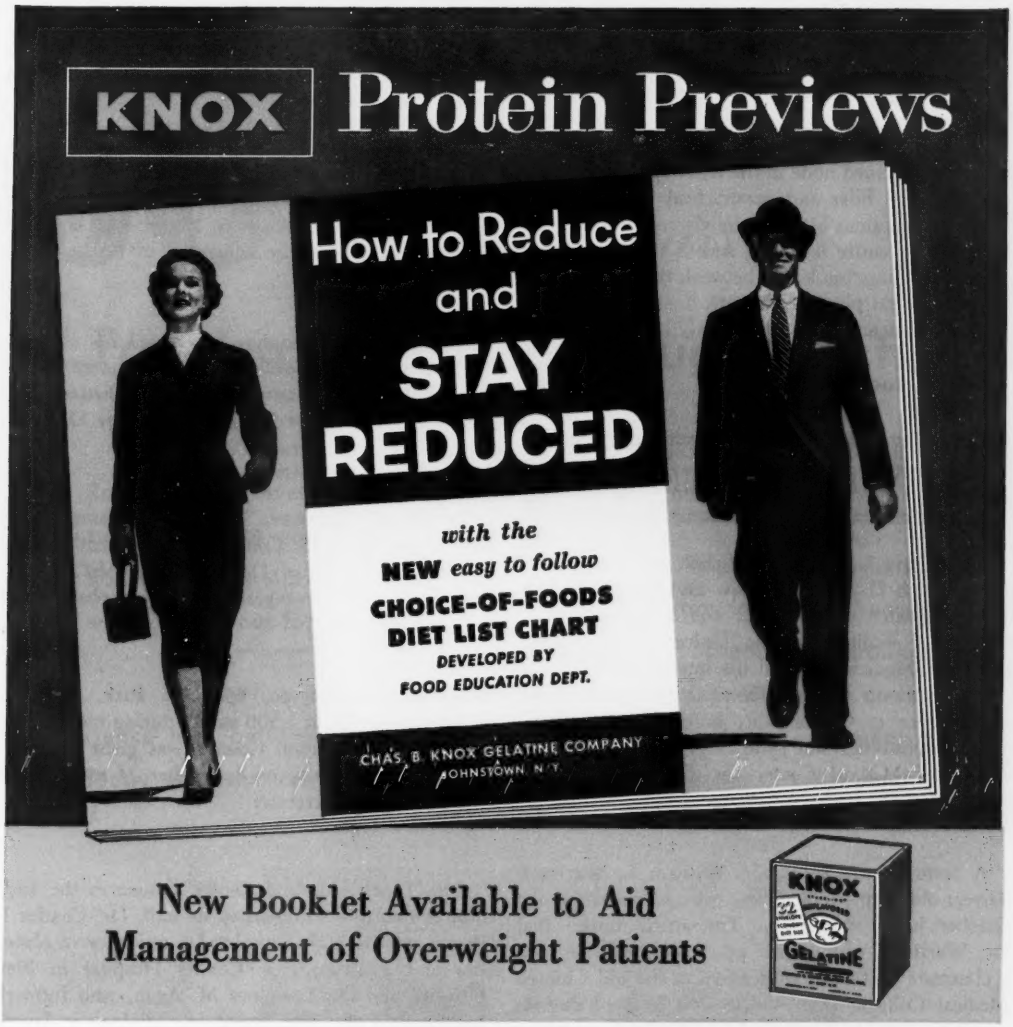
Acute passive congestion of kidneys, liver, spleen, stomach, moderate.

Bilateral hydrocele, moderate (history of considerable swelling of the scrotum a few days before death).

Acute hemorrhagic cystitis, slight.

Vacuolization of the basophilic cells of the pituitary, slight.

Patchy depletion of lipoid and tubular deformation of the glomerulosa zone of the adrenal cortex, moderate.



The 1955 edition of the well-known Knox "Eat-and-Reduce" booklet eliminates calorie counting for your obese patients. This year's edition is based on the use of Food Exchange Lists[1] which have proved so accurate in the dietary management of diabetics. These lists have been adapted to the dietary needs of patients who must lose weight.

The first 18 pages of the new booklet present in simple terms key information on the use of Food Exchanges (referred to in the book as Choices). In the center, double gatefold pages outline color-coded diets of 1200, 1600, and 1800 calories based on the Food Exchanges. Physicians will find these diets easy to revise to meet the special needs of individual patients.

To help patients persevere in their reducing plans, the last 14 pages of the new Knox booklet are devoted to more than six dozen *tested*, low-calorie recipes. Please use the coupon below to obtain copies of the new "Eat-and-Reduce" booklet for your practice.

1. Developed by the U. S. Public Health Service assisted by committees of The American Diabetes Assn., Inc. and The American Dietetic Assn.

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Professional Service Dept. SJ-10
Johnstown, N. Y.

Please send me____copies of the new illustrated Knox "Eat-and-Reduce" booklet based on Food Exchanges.

fibrosis within the nerve itself, and marked diffuse vacuolization along the nerve fibers.

Finally, we saw rather extreme degeneration and atrophy, marked by shrinkage of the fibers which appear excessively shrunken and wavy in outline. There was some interstitial fibrosis. Early interstitial invasion of fat was apparent, and a mild degree of concentration or proliferation of sarcolema nuclei was present.

These changes are characteristic of the classical picture of amyotrophic lateral sclerosis as the pathologist sees it at autopsy. I do not think that there can be reasonable doubt that the disease actually existed.

The remainder of the examination of the body was not too revealing. The lungs were studied carefully. They were relatively small, weighing only 450 grams, and hardly the lungs expected with a significant degree of cardiac failure with passive congestion. There was extensive adhesion present, marking perhaps the pneumonia that was described in the clinical history. The most dramatic changes, however, were found on microscopic examination.

Scattered throughout many sections of all lobes of both lungs were multiple remote and organizing pulmonary emboli. The results of this process, I believe, were reflected in the larger pulmonary arteries and in the heart. A section of the pulmonary artery, perhaps of the third or fourth order, showed a profound degree of sclerosis of the wall involving primarily the media and the intima with a rather extensive degree of atheromatous deposit.

The heart was not too remarkable in gross appearance. It was not heavy, weighing 350 grams. There was questionable hypertrophy of the left side, but, more significantly, the right heart appeared to be dilated and hypertrophied. The right ventricle measured seven millimeters in thickness. This is the picture that was described by Castleman and Bland and has since come to be known as the Castleman-Bland syndrome. It is a multiple embolization of the pulmonary vascular tree associated with cor pulmonale and certain classical clinical features.

We believe, in summary, that the basic cause of death in this patient was amyotrophic lateral sclerosis. We can contribute nothing to the understanding of the etiology of that process. Perhaps, however, the history does suggest the faint possibility of trauma. We do believe that the termination was related to the neurological disorder, but the fact that it was in some way mediated by this pulmonary embolization is too inviting a possibility to be completely overlooked.

Dr. Delp: Any questions of Dr. Mantz?

Wood: Did the aorta show any evidence of syphilis?

Dr. Mantz: The aorta was entirely free of such evidence. There was a minor degree of senile ectasia with atherosclerosis.

Wood: The mediastinum was not enlarged?

Dr. Mantz: The mediastinum was not significantly enlarged.

Question: Where did the emboli come from?

Dr. Mantz: That is the $64 question. We never found a source. The possibility that this was derived from the periprostatic veins cannot be overlooked. On the x-ray films, phleboliths were found in the pelvis. We were able to confirm their origin in the periprostatic areas. These may have been old thrombi which gave rise to the emboli, and the emboli were apparently of considerable age.

Nash: There was no coronary artery disease?

Dr. Mantz: Coronary involvement by atherosclerosis was minimal. There was no change in the muscle by histologic examination.

PATHOLOGICAL ANATOMICAL DIAGNOSIS

Primary

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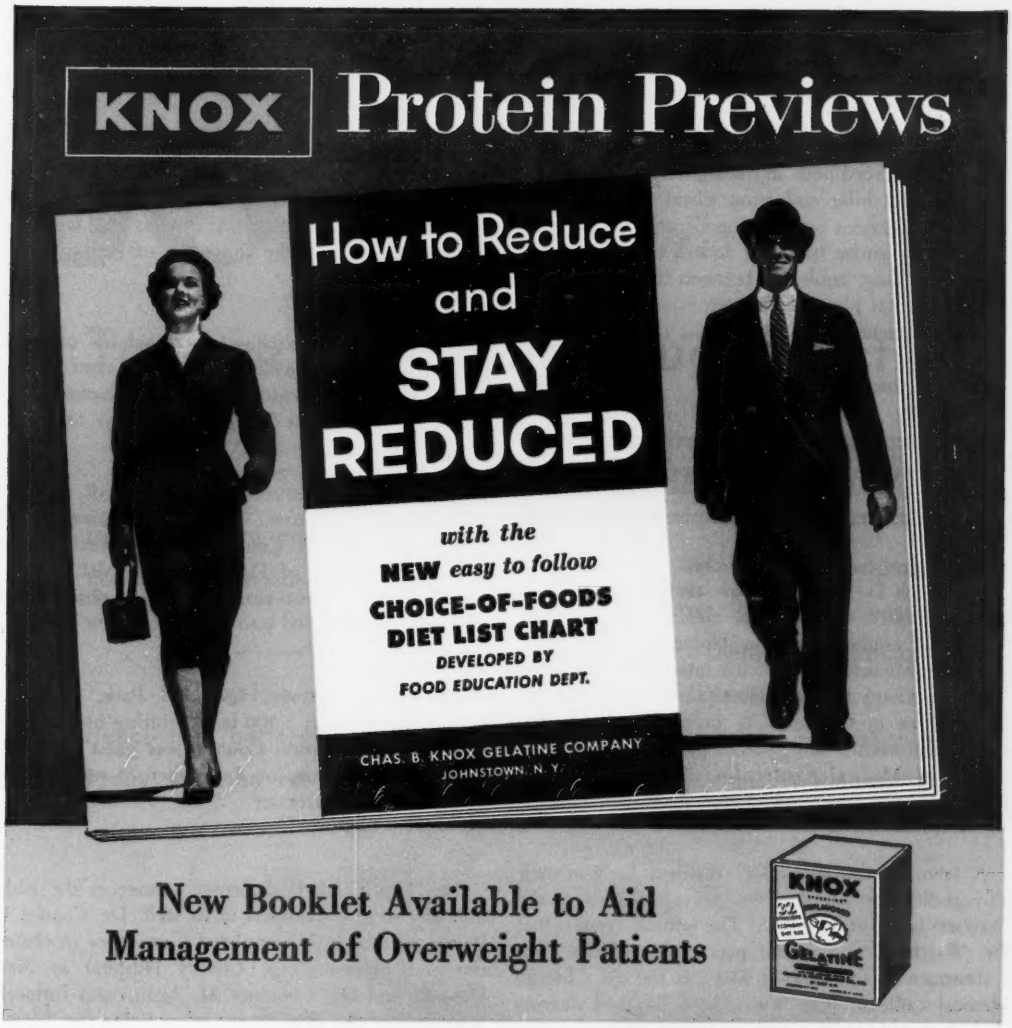

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Accessory

Arteriosclerosis of the aorta, slight.

Fatty infiltration of the myocardium and pancreas, slight.

Large calcified subpleural node in the right lower lobe, small calcified node in the right upper lobe, and partly calcified hilar and paratracheal lymph nodes.

Fibrous adhesions between the visceral and parietal pleura of the entire left lung and of the upper lobe of the right lung, moderate, between the pericardium and left parietal pleura, moderate.

Nodular adenomatous hyperplasia of the prostate, moderate.

Chronic prostatitis, moderate.

ACTIVITIES OF MEMBERS

Dr. Jerome S. Menaker, Wichita, announces that Dr. Kenneth D. Powers is now associated with him in the practice of obstetrics and gynecology. Dr. Powers, a graduate of the University of Kansas School of Medicine, served his internship at Kansas City (Missouri) General Hospital and was in general practice in Cawker City before taking a residency in obstetrics and gynecology at the University of Kansas Medical Center for three and one-half years.

A feature story about Dr. William L. Warriner, 93-year-old Topeka physician, was published in the October issue of *Coronet*. The article relates that Dr. Warriner, who is still practicing medicine, was a classmate of Dr. Charles Mayo at the old Chicago Medical College, from which both received degrees in 1888.

The Bethel Clinic, Newton, announces that Dr. Joseph J. Duerksen is now a member of its staff and will devote his time to general practice. Dr. Duerksen is a graduate of the University of Kansas School of Medicine.

Dr. William R. Lentz, a 1953 graduate of the University of Kansas School of Medicine, is now practicing in Seneca in association with Dr. Conrad M. Barnes. Dr. Lentz served his internship at Menorah Hospital, Kansas City, Missouri, and then practiced for a year in Hickman Mills, Missouri. During his student days he spent his preceptorship with Dr. Barnes in Seneca.

Dr. Farris D. Evans, Wichita, was named surgeon general of the Veterans of Foreign Wars at the national encampment of the organization held in Boston last month.

Dr. George Malouf, formerly of Greensburg and Leoti, has moved to Macksville and has taken over the practice of Dr. William A. Nixon, who is taking postgraduate work at the University of Pennsylvania.

Dr. Charles F. Haughey has closed his office in Tribune to accept a position with the Student Health Center at Kansas State College, Manhattan. The practice in Tribune is being cared for by Dr. Willard F. Werner.

Dr. M. M. Tinterow, Wichita, was named secretary of the Sedgwick County Society recently to fill the unexpired term of Dr. Charles P. McCoy, who has enrolled for a two-year residency in obstetrics in St. Louis.

Dr. R. D. Grayson, Overland Park, who has delivered more than 3,500 babies during his 33 years of practice in Johnson County, was guest of honor at a community party on September 24 marking his 65th birthday anniversary.

The Topeka Medical Center announces the addition of two new members to its staff, Dr. Charles F. Pierce, who recently completed a residency in obstetrics and gynecology at Charity Hospital in New Orleans, and Dr. Lawrence M. Agan, who formerly was head of the department of radiology at Lowell General Hospital, Lowell, Massachusetts. Dr. Pierce is a graduate of the University of Pennsylvania School of Medicine and Dr. Agan of the University of Vermont School of Medicine.

Dr. Victor G. Henry, who was stationed at Fort Leavenworth during two years of service in the Army, was released from duty last month and has returned to his practice at the Axtell Clinic, Newton.

Dr. Donald P. Trees, Wichita, disaster chairman of the Sedgwick County Society, received a certificate of honor from the American Red Cross for the society's work in the recent Udall tornado emergency.

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DEATH NOTICES

James Arthur McLaughlin, M.D.

Dr. J. A. McLaughlin, 85, an honorary member of the Sedgwick County Medical Society, died at Wichita on August 19. He had practiced more than 50 years after his graduation from Barnes Medical College in St. Louis in 1899. His first office was in Argonia. He later practiced in Norwich and in Wichita, and in 1913 he moved to Greensburg and remained there until his retirement. In 1949 he was honored by friends in Kiowa County with a celebration at Greensburg marking his 50 years of service. During World War I he served in the Army.

James Dennison Colt, Sr.

A physician who served as president of the Kansas Medical Society in 1933, Dr. James D. Colt, Sr., 88, died in Manhattan on August 31. He was an honorary member of the Riley County Medical Society, which he helped organize.

Born in Tennessee, Dr. Colt came to Kansas in 1887 to open a drug store in Riley. He later entered the University Medical College of Kansas City and received his degree in 1900. He began practice in Manhattan in 1901 and retired in 1945. His son, Dr. James D. Colt, Jr., and his grandson, Dr. James D. Colt, V, continue the practice.

Frederic E. Nipple, M.D.

Dr. F. E. Nipple, 80, who had practiced in northeastern Crawford County for almost 50 years, died at a Pittsburg hospital on September 1. He had been in poor health for two years.

Dr. Nipple received his education at the University Medical College in Kansas City, graduating in 1906, and immediately afterward opened an office in Mulberry. He continued to practice there until he entered the hospital in May.

Delos Meeker Stevens, M.D.

Dr. D. M. Stevens, 65, died at a Lawrence hospital on September 2 after an illness of two weeks. He was a native of Oskaloosa and had practiced there for 43 years, since his graduation from Kansas Medical College, Topeka, in 1912. He was an active member of the Jefferson County Medical Society.

John McGill Porter, M.D.

Dr. John M. Porter, 56, died at a Concordia hospital on September 5, exactly four months after he had taken office as president of the Kansas Medical Society. He had been active in his practice of internal medicine in Concordia and in serving the Society until two days before his death.

Dr. Porter was graduated from Harvard Medical School in 1926, served an internship at Henry Ford Hospital in Detroit, and began practice in Concordia in 1928. In 1938 and 1939 he did postgraduate work at the University of London and the London Heart Hospital.

In addition to serving the Cloud County Medical Society in many capacities, including that of president, Dr. Porter served the state organization in all of its offices. For eight years he was secretary of the Society. He then served a five-year term as one of the Kansas delegates to meetings of the A.M.A. and was successively elected second vice-president, first vice-president, and president-elect.

He had also served as president of the Golden Belt Medical Society and had held the same office in the Kansas Heart Association. During a four-year period he was a member of the Kansas State Board of Health.

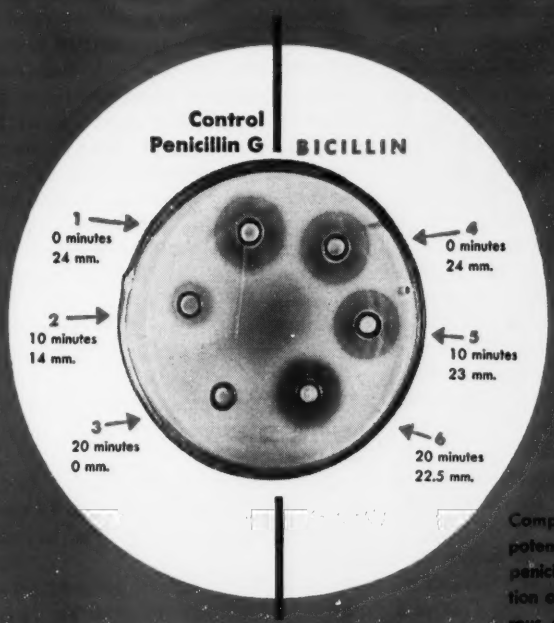
During World War I Dr. Porter was a second lieutenant in the field artillery. As a Navy physician he was on active duty for four years during World War II, receiving his discharge as a captain.

William Osee Poston, M.D.

Dr. W. O. Poston, 70, who had practiced in the Quenemo community for 42 years, died at a Topeka hospital on September 15. He was an honorary member of the Osage County Medical Society. A native of Indiana, he was graduated from the Indiana University School of Medicine in 1908 and came to Kansas the following year. He practiced for a short time in Garden City before moving to Quenemo.

Frederick Donald Smith, M.D.

Dr. F. D. Smith, 57, a member of the Sedgwick County Society, died at a Wichita hospital on September 13 after a long illness. He received his education at Notre Dame University and Northwestern University Medical School, winning his degree from the latter in 1922. He practiced first in Chicago and later came to Kansas to open an office in Belleville. In 1933 he moved to Wichita and engaged in general practice there and in Valley Center. He was active in boys' work and served as a director of Sunflower Boys' State.



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1. American Medical Association: New and Nonofficial Remedies. J. B. Lippincott Co., Philadelphia, 1954, p. 147.

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THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

Although Salk vaccine now is coming from the laboratories in encouraging volume, in Washington there still are unresolved questions that may well go beyond the problem of controlling poliomyelitis.

After months of wrangling, Congress this year enacted only one law dealing with the new vaccine. This was an authorization for the allocation of money to states to help finance inoculation campaigns. On this there was a sharp difference of opinion. Some lawmakers wanted to give federal money but to earmark it for the exclusive use of children who had passed the "means test," that is, whose parents had been officially determined to be unable to pay for the shots. Others would have nothing to do with a bill carrying the "means test."

As finally enacted, the law provides enough money to buy vaccine for only approximately one-third of all children under 20 and pregnant women. That is a concession to those who want a "means test." But the "no-means test" faction was appeased by another provision of the law, a stipulation that in inoculation programs arranged by the state and communities no financial questions could be asked.

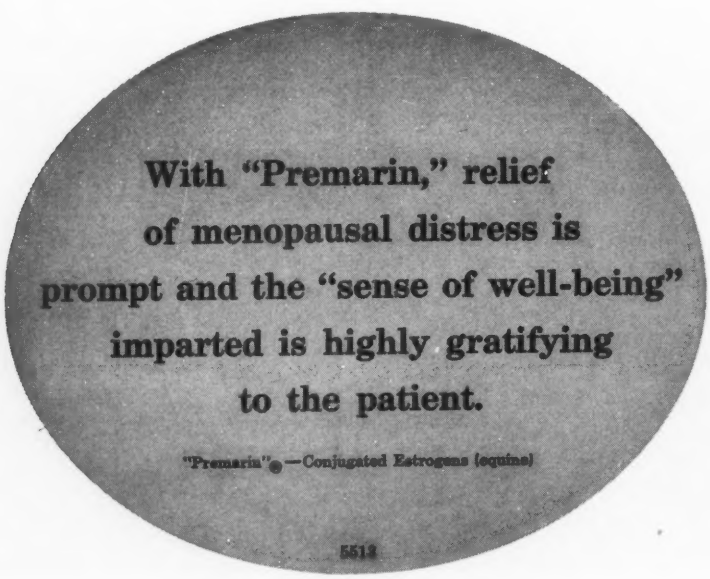
It may be that this decision will be final, that Congress will have nothing more to do with this complicated problem, except possibly to add to the 30 million dollars already appropriated to pay for vaccine. But that isn't the way some members of Congress feel. They want to reopen the entire question before the present law expires next February 15. At the very least, these senators and representatives want Congress to vote enough money to buy shots for all children in the eligible ages. In fact, those who want the federal government to play a larger role in inoculation programs regard the law now on the books as merely a temporary measure. They are looking forward to reopening the issue.

If this is done, the many questions that the last session couldn't decide again will be before Congress. Here are some of them:

1. Is it the responsibility of the federal government to make free shots available to all, regardless of ability to pay?

2. If there is to be a "means test," should the states or the federal govenment set the dividing line between the families that can pay and those that can't?

3. Should the federal government move into the picture and allocate the available vaccine, or should distribution continue along the present voluntary lines?



4. Should the states and communities arrange for all inoculations themselves?

Underlying these questions are some issues that go beyond Salk vaccine. Some persons in Congress believe there should be no limit to the participation of the federal government in public health programs. They would like to see free inoculations not only for poliomyelitis but also for all other communicable diseases for which there is a specific vaccine.

Also, the rambling system of federal control over drugs, with enforcement spread among half a dozen departments and agencies, is under criticism. Some leaders in Congress believe the whole area of federal drug control should be surveyed and possibly more clear-cut lines of enforcement laid down. One bill on this subject—which was not pressed last session—would give the Secretary of the Department of Health, Education, and Welfare authority to move in and assume control over the distribution and even the use of any drug when the Secretary decided that the public welfare warranted such drastic action.

NOTES

During the current fiscal year the U. S. will be spending a total of over 32 million dollars to help in vocational rehabilitation work, most of it in the form of grants to states.

In exchange for patent rights, colleges and laboratories will receive some financial concessions from the Atomic Energy Commission in purchase of nuclear materials and equipment.

From now on Air Force physicians, when addressed verbally, will be called "doctor." The military rank and title will continue to be used, however, in written communications.

The Department of HEW's many medical research programs are being scrutinized by a special committee set up by the National Science Foundation. In originally suggesting the study, former HEW Secretary Hobby said the time had come to re-evaluate the extent of federal medical research. Final findings will be turned over to HEW Secretary Marion B. Folsom.

JONES SCHOLARSHIP FUND

A scholarship fund honoring the late Dr. Harold H. Jones, Winfield, has been set up under the auspices of the Kansas Chapter, American College of Physicians. Contributions may be sent to either of two banks in Winfield, addressed to the attention of Mr. John Jarvis, First National Bank, or Mr. Cecil Coffey, State Bank.

Make hotel reservations now for the 1956 meeting of the Kansas Medical Society, Topeka, April 29-May 3.

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The Etiology of Diabetic Neuropathy

George E. Langsjoen, M.D.

Seattle, Washington

INTRODUCTION

The purpose of this paper is to review the literature on the etiology of diabetic neuropathy. This subject is controversial and several theories exist, some better accepted than others. No attempt will be made to review the symptomatology, physical findings, pathology, or treatment, about which there is much less controversy, and those subjects are well described in medical journals.

HISTORY

The first mention of the involvement of the nervous system in diabetes mellitus appears in John Rollo's book, *Cases of Diabetes Mellitus* (1798), in which the author notes the presence in diabetic patients of pains and paresthesias, especially in the lower extremities, as "lumbago" and "sciatica" in such a degree as to limit the use of the lower extremity.

Before 1850, the frequency of these neurological abnormalities in diabetic patients led writers to believe this disease was one of the nervous system itself. Marchal de Calvin in 1864 first recognized that diabetes might be the cause, rather than the result, of nervous disturbances.

Auche (1890) reviewed the known facts about the relationship between nervous disorders and diabetic neuropathy and observed that these did not correlate with the blood or urine sugar.

Between 1880 and 1900, the majority of the neurological complications of diabetes mellitus were clearly described.[1]

CLASSIFICATION

There have been attempts at classifying the types of diabetic neuropathies in respect to their characteristics and etiology. These will be considered since they take into consideration the possible role of general etiological factors. They also will aid in defining what we are actually concerned with in this paper.

Gregory and Lindley (1947)[2] mention Leyden's old classification into (1) hyperesthetic or neuralgic, (2) motor or paralytic, (3) ataxic or pseudotabetic forms with peripheral sensory deficiencies, absent tendon reflexes and absent pupillary reflexes. This classification lends little to our problem.

Jordon (1936)[3] proposed four types of diabetic neuropathy: (1) The hyperglycemic type which includes cases in which tenderness of nerves and muscles is the only symptom. These cases supposedly respond promptly to adequate diabetic management. (2) The circulatory type in which, besides impaired circulation, there is found to be pain, paresthesia, hyperesthesia, and hyporeflexia. (3) The degenerative type characterized by rather mild neuritic symptoms which begin insidiously and progress slowly over a period of years. (4) The neuritic group in which the onset is rather acute and improvement follows slowly with adequate diabetic management. Jordon himself believes that his groups 2 and 3 probably are identical.

Wilder[4] accepts only two real categories, the hyperglycemic and the neuritic, and believes that the circulatory and degenerative types belong to the neuritic group.

Treusch (1945)[5] brings out the point that "diabetic neuritis" is, in many cases, a misnomer. In many cases in which the condition is called "diabetic neuritis," the main etiologic factor is not diabetes per se; in other cases the condition is not truly neuritis. Since some clinicians consider pain alone adequate for the diagnosis of diabetic neuropathy, there has been a tendency for many essentially different processes to be referred to loosely by the term "diabetic neuritis." Because of this, he feels it is necessary to have classifications of diabetic neuropathy. He groups patients with "diabetic neuropathy" into three groups: (1) diabetics with pain, (2) diabetics with ischemic neuropathy, and (3) those with diabetic polyneuritis.

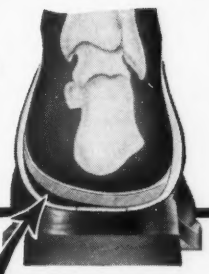
The first group includes diabetics with pain, especially in the lower extremities but also in the arms and back. This pain is worse at night. No physical signs are found other than slight tenderness. The diabetes is always out of control and responds promptly to diabetic management.

In group two, ischemic neuropathy, vascular sclerosis with arterial insufficiency of some degree is almost always present. The onset is insidious. Men are affected more frequently than women. Pains, cramps, and paresthesia, occurring especially at night, constitute the main symptoms. The principal finding is a decrease in the peripheral reflexes. Involvement is primarily of the lower extremities. In this group the disease tends to be progressive but may improve slightly if the vascular status can be improved.

Group three is true diabetic polyneuritis. There is

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Dr. Langsjoen is now interning at King County Hospital, Seattle, Washington.

Foot-so-Port Shoe Construction and its Relation to Weight Distribution



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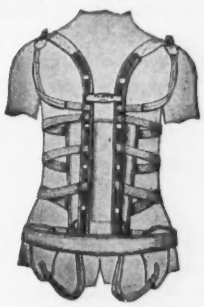

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usually an acute onset. It may occur in the young as well as the old diabetic. It may involve any part of the nervous system, but most often the lower extremities are involved. There are definite neurological symptoms and physical findings. The author states, "Here are included peripheral polyneuritis and polyradiculitis, with its secondary changes in the spinal cord. The clinical picture may be that of neuronitis and suggests the Guillain-Barre syndrome. Whereas diabetic pseudotabes presents a more or less unique clinical syndrome, it may be dependent on changes in the peripheral nerves and roots; hence its inclusion in this group may be justified."

The author adds to these a fourth group called "diabetic visceral neuritis." He refers to the peripheral vasomotor fibers of the autonomic nervous system. He adds this group mainly to emphasize that it is frequently overlooked. I believe this could be included with group three.

In the following discussion of the etiology of diabetic neuropathy, the groups Treusch called "diabetic polyneuritis" and "diabetic visceral neuritis" are those with which we will be concerned.

ETIOLOGY

Three theories have been considered at greater length than others, and these will be considered first. These three are: (1) vascular, (2) vitamin deficiency, and (3) metabolic.

I. *Vascular*

Arteriosclerotic vascular disease has already been mentioned in Treusch's second group. However, investigation of the role arteriosclerosis plays in true diabetic neuropathy has also been carried out. Diabetics are well known to be much more prone to develop arteriosclerosis than non-diabetics, and they do so much earlier in life.

Martin[1] quotes Cecil (1940) and Hines (1941) as stating that arteriosclerosis is invariably present in diabetics past the third decade of life. This led to the early suggestion that this is an important factor in the true etiology.

Woltmann and Wilder[4] (1929) studied histological sections of involved nerves and their accompanying nutrient vessels and concluded that decreased nutrition to involved nerve was a likely cause of diabetic neuropathy.

Needles and Williams[6] (1939) state that vascular changes in nutrient arteries to peripheral nerves may be responsible for diabetic neuropathy in at least some cases. They further state that in diabetics peripheral vessels and cardiac vessels are affected in 37 to 46 per cent respectively, and the brain in 2 per cent. In non-diabetics, they found cerebral and renal vessels to be involved more frequently, and they concluded the site of predilection for vascular changes is different in diabetics and non-diabetics. They could give no reason for this. They suggested a further study of nutrient vessels of peripheral nerves of diabetics and patients with generalized arteriosclerosis to ascertain the difference in quality as well as the extensiveness of lesions.

Root[7] (1946) states that the universal reversal of diabetic neuropathy in young patients makes impossible the belief that the lesion depends upon permanent degenerative changes characteristic of occlusive vascular disease.

The problem is not whether arteriosclerosis is present, but whether it is important in the production of neuritic symptoms. Martin[1] (1953) used two methods to test the state of circulation in the lower extremities: (1) skin temperatures after intravenous injections of 50 mg. of Priscoline and (2) oscillometric readings of the pulsations in the calves. These tests were carried out on 20 patients with vascular disease and diabetic neuropathy and in four patients with diabetic neuropathy without vascular disease.

Martin summarized these tests by stating that "The results of these tests do not suggest that vascular disease is of importance in the etiology of diabetic neuropathy. The latter occurs with a good circulation and is frequently absent in cases with the most severe ischemia. The sudden development of the nervous disorder, and in many cases its rapid response to adequate control of the diabetes, are additional factors against an ischemic etiology."

Karnosh (1949), unable to explain the entire picture of diabetic neuropathy on an arteriosclerotic basis, suggested that the neuropathy of young diabetics be separated from that of the elderly and thought ischemia was responsible only in older patients.

It was apparent from the literature that there have not been sufficient studies of pathological specimens to draw any conclusions in regard to arteriosclerosis being the etiological factor. It would seem that pathological studies made after death of diabetics would certainly show increasingly higher degree of arteriosclerosis of nutrient vessels since diabetics are living longer life spans than before insulin management. Beneficial studies might be possible in young diabetics with the picture of diabetic neuropathy.

II. *Vitamin Deficiency*

This leads the group of various theories in the amount of research and support given. Some authors have even maintained that this explains the entire picture.

Much of the work concerning the role of vitamin deficiency speaks mainly for the relationship between polyneuropathy and the deficiency and not necessarily between vitamin deficiency and diabetic neuropathy. Nevertheless, this was the early approach in research concerning the etiology of diabetic neuropathy.

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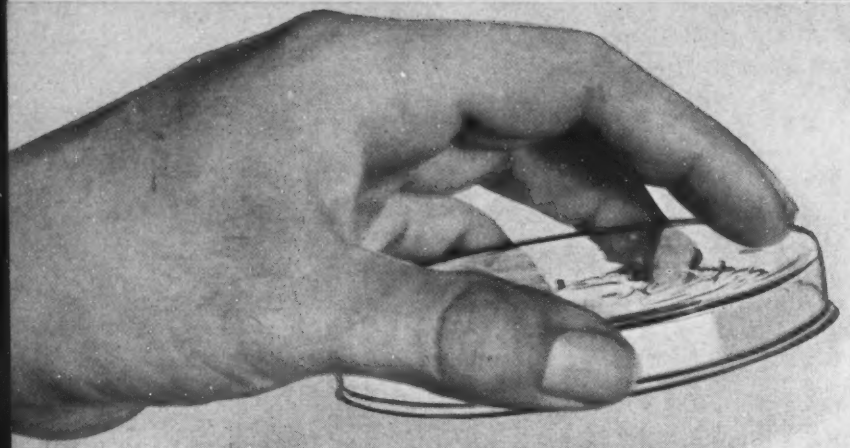

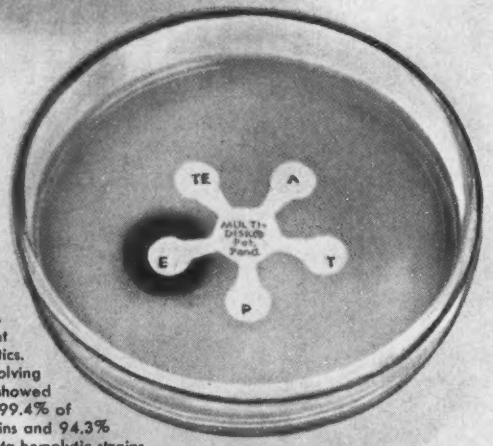

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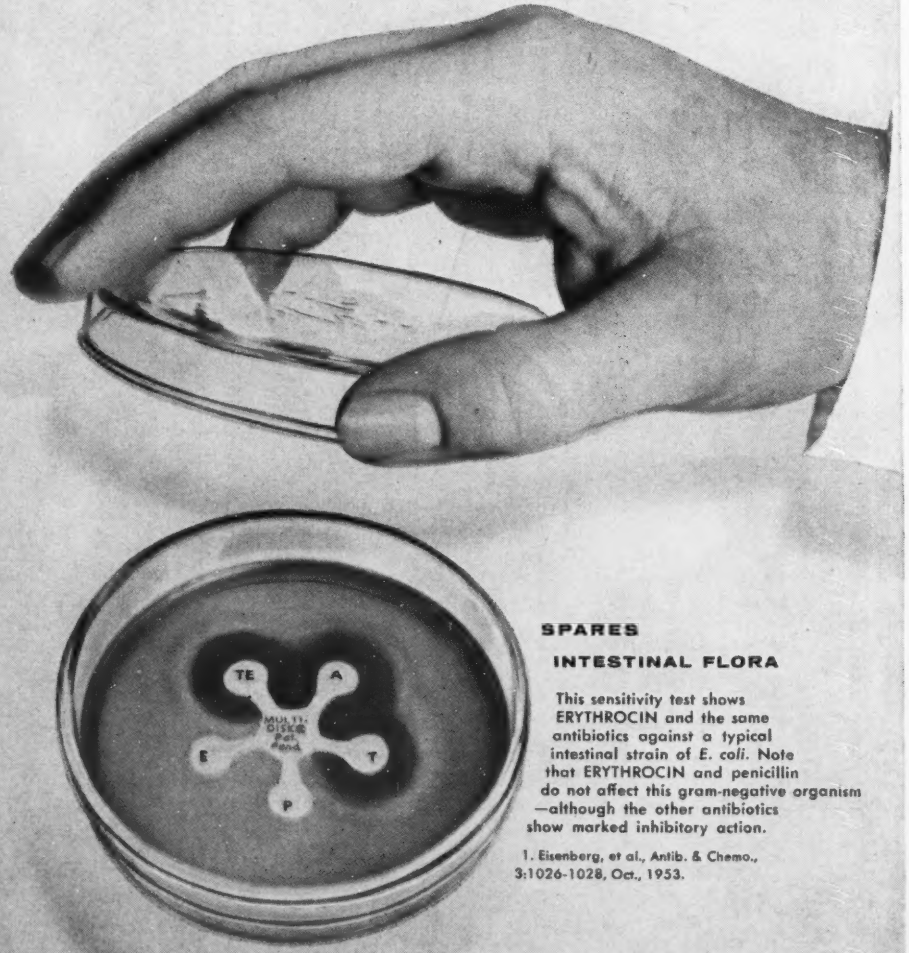

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1. Eisenberg, et al., Antib. & Chemo., 3:1026-1028, Oct., 1953.

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caused by diabetes mellitus and alcohol was remarked upon by several of the earlier writers (Auerbach, 1887; Von Leyden, 1887; Buzzard, 1890; Charcot, 1890), and in 1922 Harris suggested that they might have a common etiology. Subsequently cases were reported of diabetic patients with clear-cut deficiency syndromes and in 1926, Wohl, after investigating the nutritional state of a number of diabetics with neuritic symptoms, expressed the opinion that vitamin deficiency was the cause of the nervous disorder."[1]

Cob and Coggeshall[8] stated in 1934 that the etiology was rapidly coming to light. They believed diabetic neuropathy might be due to a dietary restriction imposed upon the patient, thus causing a vitamin deficiency.

Needles,[6] in 1939, studied three cases of diabetic neuropathy from the standpoint of a calculated vitamin-calorie ratio. In all cases, thiamine was found to be adequate. They do suggest that even though the diet is adequate in thiamine, the factors of absorption or utilization of the absorbed vitamin cannot be ignored.

Jollife et al.[9] (1940) found nine cases of diabetic neuropathy in 422 diabetics who had characteristics of peripheral neuropathy in proved thiamine deficiency. Treatment with 10 mgs. of thiamine daily without other changes in the regimen resulted in complete cure of eight patients and improvement in the ninth. Indifferent results were obtained in diabetic patients who had only vague aches and pains of lower extremities. Their conclusion was that thiamine was the etiological factor.

Meikeljohn[10] (1940), after a study of unnumbered cases, concluded thiamine definitely was not the answer, but could be, along with a total nutritional deficiency, especially carbohydrates. He was against thiamine as the sole etiological factor.

Swank[11] (1941) fed young pigeons highly purified thiamine-free diets in amounts sufficient to prevent large weight loss and found it invariably led to opisthotonus. Less deficient diets led to ataxia, leg weakness, and cardiac failure. His conclusion and contribution to the problem was that severe or total deprivation leads to death without pathological findings when a chronic subtotal deprivation led to pathological findings of a nerve degeneration.

Street[12] (1940) in his experiments with dogs confirmed Swank's conclusion. He fed dogs subtotal thiamine deprivation diets. His conclusion: inanition, accompanied by chronic thiamine deficiency can, at *most,* account for only a small share of the myeline degeneration observed.

Williams[13] (1943) was apparently the first to experiment with human volunteers. He fed two persons a 2,000 calorie diet with a daily thiamine content of 0.2 mg. per day with a test dose of 1 mg. every two weeks, raising the daily dose to 0.35 mgs. per day. First sign at the end of 30 days was a decrease in urinary excretion of thiamine. After 50 days, a test dose of 1 mg. was followed by a decrease in urinary excretion of thiamine. After this time, whenever dextrose was given, abnormally high blood pyruvic and lactic acid were encountered.

At about this same time, complaints of paresthesia and asthenia were encountered. After 110 days, polyneuropathy was apparent in both patients. Eighteen controls on the same calorie diet with thiamine furnished were observed and had no symptoms. Most of these patients responded to thiamine in several weeks and one in four months. His conclusion was that the diet must be partially deprived of thiamine to show the characteristics and pathology of neuropathy.

Stare[14] (1943) cites previous work by various men who have found other members of the vitamin B complex to cause peripheral neuropathy. Engle in 1938 incriminated riboflavin and, somewhat later, pantothenic acid. Wintrobe, in 1942, under experimental conditions, found calcium pantothenate and pyridoxine to prevent sensory neurone degeneration in growing pigs.

Rudy[15] (1945) brings up the important factor that diabetic neuropathy doesn't respond to thiamine as does thiamine deficiency neuropathy itself. He further states that diabetic neuropathy can be precipitated by complications of diabetes or by control of diabetes, whether by diet or insulin. He says there is no relationship between the duration or severity of diabetes and the resulting neuropathy, thereby contradicting the supposition that the above findings of Street, Swank, and Williams add much to the picture.

Rundles[16] (1945) states that neither occlusive vascular disease nor primary or conditional vitamin B_1 deficiency plays a definite etiological role in diabetic neuropathy.

Root[7] (1946) mentions three reasons why thiamine deficiency is often linked with diabetic neuropathy: (1) the relationship of carbohydrate metabolism to thiamine, which functions as co-enzyme in many important metabolic reactions; (2) similarity of the clinical picture of diabetic neuropathy and thiamine deficiency neuropathy, and (3) a disproportion between carbohydrate and protein calorie intake and thiamine intake as well as increased loss of thiamine in the accompanying polyuria in uncontrolled diabetics.

Root, in studying cerebrospinal fluid findings, believes the lesion in diabetic neuropathy is found chiefly in the central nervous system, whereas it is found peripherally in thiamine deficiency neuropathy. He adds that diabetic neuropathy may occur in spite of adequate thiamine intake.

He concludes by stating that the role of thiamine

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deficiency is difficult to assess without accurate measurement of carbohydrate oxidation and vitamin balance. He believed that if thiamine deficiency was important in diabetic neuropathy, it was really dependent upon the metabolic disturbance of uncontrolled diabetes.

In an editorial in *Lancet,*[17] the author concluded that in 1947 the answer was not yet known and that if vitamin B_1 is connected with diabetic neuropathy, it must be in relation to some *subtle* upset of carbohydrate metabolism.

Marble[18] (1948) only emphasizes the work of previous research in regard to the role of thiamine deficiency and diabetic neuropathy. He does emphasize, however, the factors of decreased absorption, increased utilization, increased requirement, and the increased excretion of vitamin B_1. He concluded by stating that speculation is the only possibility, at present. All his conclusions were based upon personal experience with diabetic neuropathy.

III. *Metabolic*

Many authors of recent literature conclude that the true answer to our problem lies in the faulty metabolism found in diabetes mellitus. Explanation beyond this is not extensive.

Martin states that the development of diabetic neuropathy often prolonged periods of diabetic neglect, as well as the establishment of diabetic control by the use of insulin, which suggests that the neuropathy is not only truly diabetic and intimately related to the disturbance of carbohydrate metabolism, but may, in some cases, be related to the rapidity or extent of alterations in carbohydrate metabolism and its associated changes.

DeJong[19] (1950) concludes that a failure on the part of the patient to utilize carbohydrates in amounts adequate to satisfy his metabolic requirement is followed by oxidation of fats and proteins to meet the necessary energy requirements. He believes that excessive lipid oxidation may lead to peripheral nerve demyelinization. He provides no statistics but adds that the cerebreoside, phospholipid, and cholesterol content of the involved nerves in diabetic neuropathy is much lower than that of nerves used as controls.

Martin emphasizes that although diabetic neuropathy may occur in young diabetics and those with a short history of the disease, and in well controlled diabetics, it most often occurs in chronic uncontrolled diabetics. However, he found in 112 cases that none had presented any acute complications of diabetes mellitus. In reviewing both the literature and records at King's College Hospital, he found no evidence to indicate that patients who frequently had to be admitted for acute diabetic complications of short duration were any more likely to develop neuropathy, although rapidly metabolic decompensation was present in six cases; neither ketosis nor coma appeared to be directly related to the etiology of diabetic neuropathy.

Mowry[20] (1951), in a survey of patients treated at the University of Kansas Medical Center over several years, found that 58 per cent occurred in diabetics who had marked weight loss before symptoms of diabetic neuropathy arose.

Rundles also stated that there is a striking correlation between diabetic neglect, of a degree permitting the patient to survive in a debilitating state of health without succumbing to acute complications, and the ultimate development of diabetic neuropathy. He states that there is no correlation between the severity of diabetes or any one acute complication of diabetes such as coma, acidosis, or ketonuria, and the incidence of diabetic neuropathy. He brings out the point that carbohydrate is the principal food stuff in the metabolism of nerve tissue and since the defect is an inability to oxidize carbohydrate, this may be a big point.

That ketone bodies themselves are toxic and may precipitate diabetic neuropathy has also been considered. The ketone bodies are not abnormal metabolic products and are not considered toxic or harmful to nervous or other tissues.

Rundles attempts to draw the following analogy between thiamine deficiency, which is a better known cause of peripheral neuropathy, and diabetic neuropathy. Thiamine deficiency produces a "biochemical lesion" in carbohydrate metabolism wherein there is a failure of pyruvic oxidation, with accumulation of this substance and lactic acid in the tissues. He concludes that if abnormal carbohydrate metabolism proves to be a common denominator in different types of peripheral neuropathy, it is likely unimportant with regard to the end result if the defect occurs at the 6 carbon stage, as in diabetes mellitus, or at the 3 carbon stage, as in thiamine deficiency.

Marble[18] and Root[7] too maintain that if thiamine is important in diabetic neuropathy, it is really dependent upon the metabolic disturbance of uncontrolled diabetes.

The apparent precipitation of diabetic neuropathy by initiation of diabetic management has never been adequately explained. However, persistence of diabetic control eventually leads to recovery.

Emerson[21] (1948) states that three B vitamins are as necessary as insulin itself for glucose oxidation. The patient's vitamin requirement varies directly with his insulin intake. If one increases the insulin intake, one also increases the vitamin requirement, secondary to greater glucose oxidation. This he gives as the explanation for insulin management precipitating diabetic neuropathy.

Levitt[22] (1954) believes that diabetic neuropathy may be precipitated by diabetic control when it in-

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volves marked shifts in water and electrolyte balance leading to excessive urinary losses of water soluble vitamins and temporary changes in intracellular metabolism.

Epstein[23] (1952) divides patients with diabetic neuropathy into two groups: (1) those with arteriosclerotic vascular disease who consequently have a poor prognosis and (2) those without arteriosclerotic vascular disease who have a good prognosis. These patients have experienced prolonged poor diabetic regulation. Epstein believes that prolonged poor regulation plays a role in the precipitation or aggravation of diabetic neuropathy in either type.

Levitt more or less summarizes his viewpoint by stating that one is left with the vague concept that the abnormal metabolic state found in poorly regulated diabetic patients exerts a "toxic" effect upon the nerve tissue.

IV. *Miscellaneous Groups*

There have been other proposed causes for diabetic neuropathy which have had very little support, and the literature only mentions them. Some of these are: (1) unknown toxic substances due to the diabetic condition, (2) acetonuria which has been mentioned, (3) tocopherals, (4) dehydration (of course one of the main pathological features of diabetes mellitus is cellular dehydration), (5) cachexia, and (6) hypercholesteremia.

DISCUSSION

It is evident that the first etiological factor mentioned, arteriosclerosis, cannot account for the picture of diabetic neuropathy in all patients. If diabetic neuropathy was limited to the older age group, this would be an important factor. The proposal of Karnosh, to separate the diabetic neuropathy of young from that of older diabetics, doesn't seem feasible since the clinical picture is not different in the two age groups. One might state safely that arteriosclerotic changes in nutrient arteries may play an aggravating role in older diabetics and in those refractory to diabetic management. More pathological studies would be helpful.

In consideration of vitamin deficiency, it was seen that there are completely opposing viewpoints. Some writers maintained that they obtained complete cures; others found no relief with thiamine administration. The question arises as to whether or not the latter were dealing with true diabetic neuropathies. If dietary intake, absorption, utilization, and excretion factors could be accurately determined, this faction would achieve more significance.

In view of: (1) the opposing opinions regarding this factor, (2) the knowledge that dietary thiamine content has been found to be adequate, (3) the paucity of writers who claim thiamine relieves the symptoms of diabetic neuropathy, and (4) regression of symptoms following diabetic management alone, one might consider thiamine deficiency as being a not too significant factor. The suggestion that it is intimately related to abnormal carbohydrate metabolism leaves much to be desired in so far as clarity is concerned.

It is generally agreed that diabetic neuropathy usually occurs after prolonged periods of diabetic neglect. DeJong's interesting theory that excessive oxidation of fats leads to demyelization of peripheral nerves bears further investigation. At least this is a theory which gives a more clear picture than just stating that diabetic neuropathy is due to some vague upset in carbohydrate metabolism. No conclusion was reached concerning Rundle's idea that inability to oxidize carbohydrate, which is the principal food-stuff of nerves, was a possible etiological factor.

Hyperglycemia and toxic effect of ketone bodies have been ruled out.

Levitt's hypothesis concerning diabetic neuropathy being precipitated by diabetic management appears the most attractive. However, in these cases, it would be necessary to obtain relief of symptoms by thiamine administration, which is not always possible.

CONCLUSION

1. At present, the etiology of diabetic neuropathy has not been determined.
2. It is the opinion of most writers that diabetic neuropathy occurs after prolonged periods of diabetic neglect.
3. The etiology most likely lies in some vague abnormality of carbohydrate metabolism.
4. The best treatment would seem to be prevention of occurrence of diabetic neuropathy and, after it has occurred, prompt and adequate diabetic control —both in form of insulin and in dietary and vitamin management.

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COUNTY SOCIETIES

A meeting of the Wyandotte County Society was held at the City-County Health Building, Kansas City, on September 20. Dr. Earl C. Sifers presented a paper on "Carcinoma of the Thyroid." Eight applications for membership were received.

The Sedgwick County Society opened its fall season with a tourney on September 30 at Rolling Hills Country Club and the Wichita Gun Club. The day was concluded with a dinner at the golf club and the awarding of prizes.

An attorney, Mr. L. M. Cornish, Jr., addressed the Shawnee County Society at a meeting held at the Medical Society Building, Topeka, on September 6. He spoke on services of interest to the physician, such as Kansas Children's Home and Service League, legal aid, vocational services, facilities for outpatient psychiatric and psychological counseling, alcoholism, etc.

ANNOUNCEMENTS

The 23rd annual assembly of the Omaha Mid-West Clinical Society will be held at Hotel Paxton, Omaha, October 24-27. The program will feature 11 nationally known guest speakers, 36 lectures by members of the faculty of the University of Nebraska College of Medicine and Creighton University School of Medicine, four panel discussions, informal discussions following luncheons and dinners, scientific motion pictures and exhibits, and technical exhibits.

A registration fee of $7.50 will be charged. The assembly has been approved for credit by the American Academy of General Practice for 37 hours of postgraduate instruction.

Information may be secured from James J. O'Neil, M.D., Director of Clinics, 1031 Medical Arts Building, Omaha.

The Oklahoma Division of the American Cancer Society will present its third annual scientific session at the Huckins Hotel, Oklahoma City, on December 2. Guest speakers known throughout the nation will present the program, concerned entirely with cancer. Information may be secured from the Oklahoma Division, 937 Commerce Exchange Building, Oklahoma City 2, Oklahoma.

The trustees of the Caleb Fiske Fund of the Rhode Island Medical Society are offering a cash prize of $350 for the best essay submitted on "Use of Radioactive Isotopes in the Treatment and Investigation of Disease." The manuscripts must be typewritten, double spaced, and should not exceed 10,000 words. Deadline for entries is February 2, 1956. Information may be secured from John E. Farrell, Sc.D., 106 Francis Street, Providence 3, Rhode Island.

The next scheduled examination (Part I), written examination and review of case histories, for candidates for certification by the American Board of Obstetrics and Gynecology will be held in various cities on Friday, February 3, 1956. Current bulletins may be obtained from Dr. Robert L. Faulkner, 2105 Adelbert Road, Cleveland 6, Ohio.

The American Regents of the International Board of Proctology announce plans for international certification of two classes of applicants: (1) those desiring certification for the practice of proctology, either anorectal or anorectal and coloproctology, and (2) those seeking certification for proctology within general surgery. Those in the first category must practice proctology only. In the second group are general surgeons whose activities are partially limited to proctology. For the present there will be no certification of candidates in the United States.

Applications may be secured from the American Regents, 147-41 Sanford Avenue, Flushing, New York.

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BOOK REVIEWS

Feeling No Pain. By Bill O'Malley. Published by Prentice-Hall, Inc., New York City. 88 cartoons. Price $1.95.

Bill O'Malley's ability to find a laugh in almost any form of human activity is turned toward the hospital in his latest book of cartoons, *Feeling No Pain,* published last month. The same humor which typified a former product of his drawing board, *Blessed Event,* is apparent.

Although the patient is the subject in most of the cartoons, the physician will find himself or his colleagues in some of the good humored drawings. And he'll probably come forth with chuckles as he turns the pages.

Fluoroscopy in Diagnostic Roentgenology. By Otto Deutschberger, M.D. Published by W. B. Saunders Company, Philadelphia. 771 pages, 888 illustrations, 523 figures. Price $22.

This work seems to be, as is stated in the preface, the first attempt to compile all essential knowledge about fluoroscopy in a single volume. The author has attained a considerable degree of success and deserves thanks and congratulations for the fine organization and excellent presentation of his material.

The potentials and limitations of this diagnostic procedure are well treated. There is a concise discussion of protection against radiation hazards, technical factors, and the image intensifier in the first section. The second or clinical section is divided according to anatomical regions into the head, neck, chest, abdomen, and extremities. An appendix contains forms for reporting fluoroscopy, which also may serve as an excellent check list for the trainee or occasional fluoroscopist.

Numerous illustrations represent a valuable addition to the text. The part on the segments of the lung could well have been amplified. The correlation of radiographic techniques is indicated, and many roentgenograms are included. The format and illustrations are of the usual Saunders excellent quality.—*K.Y.*

Meeting of Pediatricians

Pediatricians of Kansas met at Emporia on September 10 for a scientific program and business session. Dr. David R. Davis, Emporia, was elected to preside over the Kansas State Pediatric Society during the coming year, with Dr. Charles T. Hinshaw, Wichita, as vice-president and Dr. Theodore E. Young, Winfield, as secretary-treasurer.

Ciba Pharmaceutical Products will soon publish the first issue of an eight-page tabloid-sized newspaper carrying reports on medical developments, activities of professional and scientific organizations, hospitals, research institutions, medical colleges and federal agencies. The publication will be distributed to physicians exclusively and will be issued every other week.

Science Contract Awards

Award of 175 unclassified life science research contracts in the fields of biology, medicine, biophysics, and radiation instrumentation was announced last month by the Atomic Energy Commission. Kansas institutions were awarded four contracts.

The University of Kansas School of Medicine, with Dr. Robert E. Stowell as investigator, will study effects of ischemia, irradiation, and chemical irritation on cytochemistry of mammalian tissues. The University of Kansas will center its work on deposition and excretion of boneseeking radioisotopes, with Dr. Frank E. Hoecker as investigator.

Two workers at Kansas State College, Dr. C. C. Roan and Dr. R. E. Clegg, will supervise separate projects. Dr. Roan will study use of radioactive tracers in investigations of the mode of action of insecticides with emphasis on potential systematic or chemotherapeutic action. Dr. Clegg's work will center around phosphoproteins of the embryonated egg.

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Historical Material Needed

In preparation for the observance of The Kansas Medical Society's centennial anniversary, members of the Committee on History are attempting to collect all material of historical interest. Physicians who can contribute information, records, etc., are urged to send such to

Committee on History
Kansas Medical Society
315 West 4th Street
Topeka, Kansas

● ● ●

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Index to Advertisers

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Ayerst Laboratories 580

Baker Laboratories, Inc. 573
Brown and Williamson Tobacco Corporation XVIII
Burroughs-Wellcome and Company VIII

Chicago Medical Society XVI
Ciba Pharmaceutical Products, Inc. 585
Coca-Cola Company IX
Corn Products Sales Company (Karo) XIII

Fairmount Hospital XIX
Foot-so-Port Shoe Company 583

Goetze Niemer Company 596

Hanicke, P. W., Manufacturing Company 583

Isle, W. E., Company 598

Kansas Oxygen, Inc. VII
Knox, Charles B., Gelatine Company, Inc. 575

Lakeside Laboratories V
Lattimore-Fink Laboratories 589
Lederle Laboratories Division, American Cyanamid Company ..... 562 and 563
Lilly, Eli, and Company XXII

Mead Johnson and Company ..... Back Cover
Medical Protective Company 595
Menninger Foundation XVI
Merchants Finance Corporation, Inc. 589
Munns Medical Supply Company, Inc. 597

Neurological Hospital XIX

Parke, Davis and Company ..... Inside front cover and III
Petro's Surgical Appliances XIX
Pfizer Laboratories, Division of Charles Pfizer and Company, Inc. ..... XVII and Inside back cover
Physicians Casualty Association 598

Ralph Clinic 577

Schering Corporation ..... XX and XXI
Searle, G. D., and Company 571
Seven-Up Bottling Company, Inc. 591
Sharp and Dohme, Inc. ..... IX, XII, XIV, XIX, 581, 589, 593 and 595
Southwest Scientific Corporation XIX

University of Colorado Department of Medicine 595
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Winthrop-Stearns, Inc. XI
Woodcroft Hospital XIV
Wyeth, Inc. ..... XV and 579

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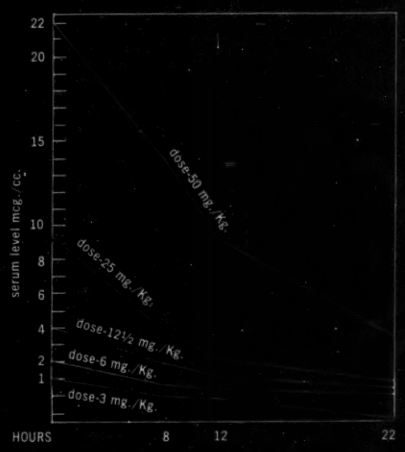

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(1) Jeans, P. C., in A. M. A. Handbook of Nutrition, ed. 2, Philadelphia, Blakiston, 1951, pp. 275-278.

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